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OF THE

American Academy of Ophthalmology and Oto-Laryngology

HELD AT

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OCTOBER 27-31, 1930

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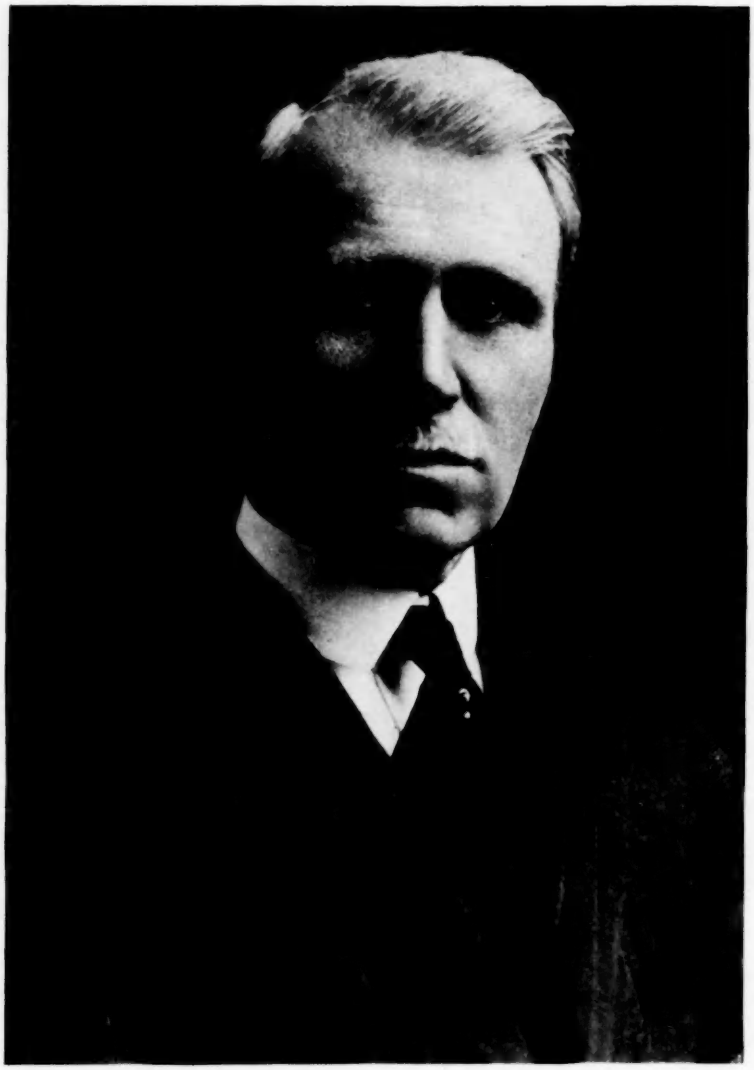

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JOINT SESSION

PRESIDENT'S ADDRESS.

WILLIAM H. WILDER, M. D.,

CHICAGO.

My first pleasant duty on opening the thirty-fifth annual meeting of the American Academy of Ophthalmology and Oto-Laryngology is to express my sincere appreciation of the honor the Academy has seen fit to bestow upon me by selecting me to preside over its affairs and deliberations for this year. The long line of distinguished and devoted officers who have preceded me have by their zeal and unsparing efforts brought this Society to its present high plane, and have set standards of work and efficiency that tax their successors to equal.

The work of preparing the program of papers and discussions that has been done by the Program Committee, including the Secretaries of the Sections, will speak for itself and, we hope, will meet with your approval.

I call your attention to the growth of the instructional program which this year consists of one hundred and eighty conferences conducted by seventy-one instructors.

In addition to this are three special courses: One on histopathology of the eye, one on histopathology of the nose and throat, and one on the writing of medical papers.

This instructional work is one of the most outstanding and most praiseworthy features of the Academy, not alone for the instruction given but for the valuable inspiration it has been to physicians throughout the country who have been able to avail themselves of its opportunities. Great praise is due Dr. Gradle and his co-workers on the committee and to the numerous instructors by whose efforts this splendid work has been made possible.

During my incumbency of office I have had the opportunity of observing the indefatigable work of our executive secretary, Dr. Wherry, to whom all of us should feel deeply indebted. Without such untiring efforts as he has shown, this Academy could not properly function.

I should like also to take this opportunity of thanking the various local committees whose devoted efforts for your comfort

and entertainment will, I trust, be rewarded by a very successful meeting.

My sad duty is to call your attention to the unusually long list of Fellows of the Academy who have departed since our last meeting. Were it not that the programs had already been printed the roll would have included the name of Dr. Ross Hall Skillern, our esteemed President of 1927. One other ex-President, Dr. William L. Dayton, is on the list, and many other distinguished ones in our profession. Their deeds will live after them, while they have gone to that undiscovered country from whose bourne no traveler returns. May I ask that you stand with me and in a brief period of silent thought pay homage to their memory?

I shall not tire you with a lengthy address, knowing that you will be much more interested in the program that has been prepared and which you should have time to hear and to discuss. On this occasion, however, I should like briefly to call your attention to a subject the importance of which should particularly interest the ophthalmologists of our Society and, by suggestion, the otologists. It is the need of attempting to unify or standardize our measurements of vision and our methods of recording them.

Valuable as are the objective examinations of the eye both external and internal, we must place great stress upon the functional examination, and this from the very nature of the case must be a subjective one, with all the chances of inaccuracy and error that belong to subjective methods of learning the truth. The subject himself must give the needed information.

In making a functional examination of the eye we test the vision to determine its light sense, its color sense and its form sense. All of these are important and in most cases are co-related, but it is the form sense that presents some of the most striking phenomena of sight and is, therefore, the one that is usually first tested. Furthermore, it is the central visual acuity that gives the most striking manifestation of sight, and an impairment of central vision brings the patient to the physician more promptly than does a much greater defect of the peripheral vision of which he may not be aware.

It is supposed that the rods in the percipient layer of the retina play the dominant rôle in the light sense, while the cones control the form sense. Certain it is that the form sense is most acute

in the region of the fovea and the macula, where the cones are massed together almost to the exclusion of the rods.

If light from two distant luminous points enters the eye, it is supposed that if the rays from these points strike separate cones, or probably alternate cones because of irradiation, the two points will appear separate.

The larger the angle between the rays, measured at the nodal point of the eye (a point near the posterior part of the lens on the principal axis), the farther apart will appear the points of light. The smaller the angle the nearer together the points will appear. This is known as the visual angle and is equal to the angle drawn from the stimulated cones to the nodal point in the lens.

The normal eye is supposed to recognize two luminous points as separate if the rays from them subtend an angle of one minute of arc (1″)—i. e., one-sixtieth of a degree. Some eyes may perceive the two points as separate at a smaller angle, even as low as forty or thirty-five seconds (40″ 35″), while some normal eyes require a larger angle than one minute. This is known as the "minimum separabile," and an angle of one minute has been pretty generally assumed to be an average.

Based on this principle, Snellen constructed his charts, using Roman letters of square Gothic form, each limb or detail of which subtended an angle of one minute of arc and the whole letter five minutes at a given distance. The letters were graduated in sizes at certain intervals, from the largest, which should be seen at 60 m. down to 6 m., or even 5 m., each letter at the given distance supposedly subtending an angle of five minutes.

In testing with such a chart, as it is impractical to place the patient at these various distances, he is kept at a fixed distance of 6 m. or 5 m., for at these distances the degree of divergence of the light rays is so slight that any effort of accommodation of the eye would be practically negligible.

The degree of visual acuity is then recorded by a conventional fraction, of which the numerator is the distance of the patient from the chart and the denominator the line read.

$$\text{e. g., } \frac{6}{5}\ \frac{6}{6}\ \frac{6}{9}\ \frac{6}{18}\ \frac{6}{60} \text{ etc., according to a formula } V = \frac{d}{D}$$

in which d = fixed distance

D = line read

Such fractions should not be reduced, as the terms represent the conditions of the test.

For illiterates Arabic figures were employed, or square figures, one side of which was missing, or the illiterate E.

It was observed that certain letters are seen more readily than others and therefore the test with letters, practical as it is, gave uncertain results. Arabic figures, proposed as a modification, are even more uncertain, although possessing certain advantages as an international test.

Various modifications have been proposed by different observers. Wolfberg suggested the use of groups of dots, each of which and the space between them should subtend a visual angle of 1 minute.

Seitz, Reber and others prepared charts with pictures of familiar objects for testing young children and illiterates, the size based on the visual angle of one minute.

Landolt suggested a figure of a broken ring, printed in black on a white background, the break in the continuity of the ring being equal to the width of the limb of the ring, which is one-fifth of the diameter of the figure. The visual acuity was expressed in the maximum distance at which this break could be perceived, a visual angle of one minute, as in the case of Snellen and other charts, being the standard of comparison. The result could be expressed either in decimals or by the conventional fraction $V = \frac{d}{D}$ (in which d = distance from the chart; D (the line deciphered.

In our country many charts are in use with the distances indicated in feet, some are expressed in meters and in varying intervals of distance and in various methods of notation, so that one reads of records of visual acuity such as $\frac{6}{20}$, $\frac{5}{30}$ 0.3 0.2 $\frac{12}{60}$, $\frac{10}{200}$ and so on.

Furthermore, there is no assurance that our charts are correctly printed, even assuming that the standard of one minute visual angle is the one most nearly correct.

It is apparent that we need to bring about some uniformity or standardization of our measurements of visual acuity and of our methods of recording them.

An attempt to accomplish this was made in 1909, when a committee of seven, under the chairmanship of Prof. Hess, presented a unanimous report to the International Congress of Ophthalmology, meeting that year in Naples.

The committee favored a chart containing the broken ring of Landolt in graduated sizes on the minute angle basis. They also favored the decimal system of notation, 1 (unity) representing normal, the different intervals of the chart being indicated in decimal tenths in an arithmetic progression—i. e., 0.9, 0.8, 0.7, 0.6, et seq. Charts made up of Arabic figures, notated in the same way were to be used. It was thought this would settle the matter of testing the central visual acuity, but the subject came up again last year at the meeting of the International Congress of Ophthalmology, at Amsterdam, in reports made by Professor Dufour of Nancy and Professor Elschnig of Prague, a committee appointed to consider the question of standardization of visual acuity.

Both agree on the one minute angle as giving a fair average of the "minimum separabile." Prof. Dufour in his report practically reiterates the conclusions arrived at by the committee of the International Congress at Naples. Prof. Elschnig dissents from this as to the form of optotypes used, preferring Roman capital letters for ordinary use and the open square □ of Snellen for illiterates.

He also prefers for the expression of the degree of vision the metric notation based on the formula $V = \frac{d}{D}$ in which d = fixed distance of patient from chart 5 or 6 meters, and D the distance at which a given line should be read—e. g., $\frac{6}{12}$ $\frac{6}{18}$, etc.

Both agree that the optotypes should be dull black on a non-glistening pure white background and the chart, whether a transparency or a card, should have a definite illumination (250 lux, according to Elschnig).

To avoid sharp contrasts and to favor light adaptation, the charts are to be placed on a background, also illuminated, of gray or neutral tint (one of the gray papers of the scale of William Ostwald, according to Dufour).

It is evident we have not yet arrived at the most exact and the most practical way of measuring the visual acuity and of recording the results.

From the very nature of the complex problem, it is probably impossible to devise a scientifically exact standard of normal visual acuity, and, as for the abnormal, any expression of the degree of visual disability must be purely relative, even though due regard has been paid to the fields, light adaptations and other complexities of the problem.

The one minute visual angle to represent the "minimum separabile" does not indicate the normal for all eyes, as is conceded, but it may be the best average.

But we should be able to agree upon and establish some standards of measurement and notation in order that our records may show more uniformity.

The American Committee on Optics and Visual Physiology, which has three representatives from this Academy, has this subject of visual acuity under consideration at the present time, as will be shown in their report; and it is their purpose to engage in some investigative work looking to the establishment of standards of observation and measurement that will be practical and as nearly as possible physiologically correct. It is to be hoped that the Academy will endorse their efforts and will render them reasonable financial aid if that should be necessary.

If agreements can be reached on suitable optotypes and measurements, it is hoped that some central authority, such as the National Bureau of Standards, will be willing to supervise and approve the manufacture of standard charts with instructions for their use.

This would be a decided improvement on the uncertain and irregular system that is now in use.

SYMPOSIUM ON DISEASES OF THE PARANASAL SINUSES AND THEIR RELATION TO DISORDERS OF THE EYE.

THE ETIOLOGY OF RETROBULBAR OPTIC NEURITIS

(ADDRESS OF THE GUEST OF HONOR).

PROF. DR. EMILE DE GRÓSZ,

UNIVERSITY OF BUDAPEST.

I feel deeply affected by your great kindness, and desire to give my special thanks for the honor that was granted to me through the kind invitation of the Academy of Ophthalmology and Otolaryngology, in affording an opportunity to speak to my distinguished audience.

I have to begin with an apology, as I cannot help feeling how moderate my abilities are, how limited my knowledge of English compared to the task I undertook in being the humble representative of a small nation, in speaking to such eminent doctors and scientists of a great and powerful country.

I must aver how many scruples I had about selecting an appropriate subject.

I gave special consideration to the suggestions of our illustrious president, Dr. Wilder. I also reckoned with the fact that a considerable number of otolaryngologists was going to attend this meeting, and finally decided to take up a study of the etiology of retrobulbar optic neuritis. I was fully aware of the effort with which ophthalmologists, otolaryngologists and neurologists had joined their forces in order to discover new stores in the inexhaustible mines of science. I have to confess my timidity as to whether I shall be able to appeal to your interests, and whether I will have the power to contribute to the valuable literature on this subject published by such distinguished scientists of your country. Will you not be justified in accusing me of having brought owls into Athens: Τλαῦκας Ἀθήναζε.

And still, in the perusal of the extensive literature dealing with this question, one is bound to confess that we are far from having

a definite and clear comprehension as to the character of this disease. However numerous the writings and lectures delivered on this subject may be, we still lack any sameness of conception and a conflicting and wide divergence of opinion prevails on this matter.

I was encouraged in my attempt to unravel this much discussed perplexity by the fact that Prof. Onodi, my well-known compatriot, had made a brilliant study of the anatomic relationship between the accessory sinuses and the optic nerve. Prof. Márk Paunz's important discoveries were also observed in the abundant material offered to him in the Ist University Eye Hospital of Budapest which attends, under my direction, an annual number of 27,000 new patients. For many decades I endeavored to find out the true relationship between the paranasal sinuses and the affections of the eye, and I made a special attempt in my publications to put the whole problem on a sound basis. In the last 25 years, since the direction of the above mentioned Eye Hospital was handed over to me, 564,983 new patients applied for eye examinations, and 44,038 of them were admitted to our wards. Thus I had an opportunity of observing affections of many hundreds of eyes during that period that proved to be of nasal origin. Special assistance was lent to me by the kind collaboration of Profs. Onodi and Paunz. We observed approximately 100 retrobulbar neuritises every year, 60 per cent of which were due to the excessive use of alcohol or tobacco, while 15 per cent proved to have a nasal cause.

My aim in this lecture is to add to the understanding of relationships based much more on my own experience than on the material available in literature.

The early diagnosis of defects located in the retrobulbar section of the optic nerve must depend upon mere functional examinations.

A central color scotoma progressing to an absolute scotoma, associated with an intact fundus, is the first symptom corresponding to the disturbances of the retrobulbar section, viz., the papillomacular set of the optic nerve.

Van der Hoeve called attention to the enlargement of the blind spot as an important sign indicating the disease. Irene Markbreiter, an assistant to Prof. Onodi, found this symptom associated in most cases of sinusitis (70 per cent). This symptom is a subject of frequent controversy, called forth by the great dif-

ficulties involved in its examination, requiring much patience on the part of the doctor, and keen attention as well as intelligence on the part of the patient, both characteristics known to be lacking in many instances.

Marked ophthalmoscopic changes appear in a more advanced stage of the disease: the inner margin of the disc becomes indistinct, filminess appears, the external part is pale, and gradual changes lead to a partial atrophy of the optic nerve.

The disease often has an acute beginning, and runs a very rapid course; form sense might disappear as well as light sense. In other instances the beginning is chronic, the progress of the disease is slow, the gradual decline of vision to finger counting takes several weeks, while the function of the peripheric field remains intact.

Improvement takes place in periods varying from but a few days to several weeks, or even months, and many cases are on record which ended with a permanently deficient sight.

The most important factors in the etiology of acute retrobulbar optic neuritis are the affections of the paranasal sinuses and multiple sclerosis, while among the causes of the chronic form of the disease, excess of alcohol and nicotine are of primary importance, though it has been known to follow diabetes, lues, lead poisoning and other intoxications. Heredity may also have a rôle in the etiology, as it was first discovered by Leber more than 50 years ago and described with such accuracy that his keen observations and his data are still most valuable with regard to this form of the disease.

Joseph Jacob Plenck, professor of ophthalmology at the University of Buda, called attention to the nasal origin of defective vision, at the end of the eighteenth century, by his statement that "Amaurosis a morbo sinus frontalis noscitur dolore in sinu frontalis haerente aut alio sinus vitio curatio exigit amotionem causae a sinus frontali" (see Josephi Jacobi Plenck, Professori Regii Publici ac Ordinarii in Regia Universitate Budensi. Doctrina de Morbis Oculorum, 1783. Editio Secunda).

We find the following chapter in the book (Lehre von den Augenkrankheiten, Wien, 1817, II, 530), published by J. Beer: "About the vicarious amaurosis of a suppressed cold, lacking any noticeable retention of mucus in the frontal sinuses." The relationship between nasal inflammatory processes and diminution of sight is shown too in this chapter: "One recognition of this

amaurosis is made uncommonly easy by means of a careful study of the history of this disease, by considering a former serious attack of cold suddenly suppressed as an important cause of eye disturbances."

In spite of these publications, many years elapsed before Berger and Tyrmann, in 1886, Zuckerkandl, in 1893, and especially the anatomic investigations of Onodi, recently revealed the connection between affections of the paranasal sinuses and those of the optic nerve. The appearance of Onodi's Atlas, published with the support of the Hungarian Academy of Science, gave rise to a particularly vivid and valuable literary discussion with regard to this question. Many overestimated the importance of nasal disturbances in the etiology of retrobulbar neuritis. This exaggeration again led to skepticism. The time has come to put this much disputed relationship on a sound basis.

The connection is obvious in many instances. Unilateral supraorbital neuralgia consequent on an attack of cold or epidemic influenza is always accompanied by tenderness of the frontal bone above the sinus, even upon a very slight touch, and pain is complained of on moderate pressure of the inner upper wall of the orbit on the pressure of the globe backwards into the orbit. Sight is reduced, and the examination of the visual field reveals central scotoma, while the periphery is unimpaired. All these symptoms speak for the fact that the disturbance of the accessory sinuses affected the optic nerve. The course of the disease reaffirms our diagnosis, as the application of camomile compresses, the exposure to camomile steam or salicylates given internally, are followed by an improvement which may result in a complete recovery within from two to three weeks. Such relationship is most likely existing in cases in which the progress of the disease is less rapid, but a discharge of pus is present in the nasal passages, and the X-ray picture reveals shaded accessory sinuses. Cure is obtained in these cases either by plugging the nose for from two to three hours with adrenalin cocain mixture or by clearing away the pathologic secretions of the affected mucous membranes.

The relationship is equally indisputable in cases in which symptoms of retrobulbar neuritis follow either an operation on the nose or the simple plugging of the nose. The optic nerve may be hurt by an accidental fracture of the bone, brought about

by chiseling at operation or it may be damaged by retention in plugging of the nose.

Uncertainty in etiology begins to take place when the only symptoms, the gradual diminution of vision and central scotoma, are accompanied by negative rhinologic discoveries and a normal disc. Many are inclined to attribute a nasal origin to these disturbances if improvement occurs in spite of negative findings revealed with intranasal examination. Others deny this view and insist that other causes must be responsible for this form of the disease. Multiple sclerosis is found to be one of these causes. There can be no doubt that this serious disease of the central nervous system is most probably of an infectious character, and is a more frequent etiologic factor of retrobulbar neuritis than it used to be considered. Diminution of vision occurs sometimes before any other symptoms set in, and general symptoms of disseminated sclerosis, such as intentional tremor, eye muscle paralysis, scanning speech and impaired reflexes, appear after a lapse of months or several years.

Scherer recorded 203 cases of retrobulbar neuritis among 64,200 patients examined in the University Eye Hospital of Tübingen, within the period of 1921 to 1928. According to his statement, 71 per cent of those had multiple sclerosis as the cause of their eye affection. He blames sclerosis also for cases presenting unknown etiology. Others, on the contrary, attribute a nasal origin to 70 per cent of the cases.

I found 58 cases of retrobulbar neuritis due to nasal etiology among the 18,587 patients examined in 1913. On the other hand, Heine found only three among 50,000 patients. The truth must be somewhere in between, as usually happens in such cases.

The connection of nasal disease and retrobulbar neuritis is difficult to be detected, as their pathologic and histologic investigation is of rare occurrence. Considering that sinusitises are frequent, those associated with retrobulbar neuritis are very rare, and it is hard to form a clear comprehension of their relations. Brückner recorded 360 autopsies revealing 165 diseased accessory sinuses, among them 95 empyemata. Nasal etiology is very uncertain in cases in which, in spite of recovery consequent to nasal treatment, symptoms of disseminated sclerosis appear in a later period. Such cases are subject to individual and subjective differences in views and opinions. These differences were manifested at the Viennese meeting of the German Ophthalmological

Society, held in 1921, when Meller, Hirsch, Réthi and others attributed primary importance to nasal factors, while Hajek, Schlesinger and others held contrary opinions. Hajek was against operative treatment if rhinologic findings proved to be negative, while Meller stood for it on the basis of mere ophthalmologic examinations. Hajek obtained improved vision by means of opening the seemingly normal ethmoid cells of one side. But he observed the same improvement without any surgical intervention when the affection of the other eye set in.

In other instances retrobulbar neuritises were thought to have recovered by means of nasal operation and later they were revealed to be due to multiple sclerosis. On the other hand, Meller blames the negligence of operative treatment for many incurable optic nerve atrophies. The discussions at the meetings of the Hungarian Ophthalmological Society, held last April, revealed the great difficulties associated with this question.

The only means to come to a proper understanding of etiologic factors is a carefully combined simultaneous ophthalmological, rhinological and neurological examination of each case, and the coordination of the various findings obtained. The marvelous results of such a collaboration are already known in this country. It is enough to refer to the achievements of Harvey Cushing and his collaborators, or to those of the Mayo Clinic. Nasal origin cannot be denied after the fundamental findings of Onodi. As the projected pictures will show, his anatomical findings are very enlightening. As this slide shows, the optic nerve is separated from the sphenoidal sinus and from the ethmoidal cells by a very thin bony wall, and there are dehiscences on that wall, through which affections might be transmitted to the optic nerve. That is why 60 per cent of the orbital inflammations are due to sinus troubles. Birch-Hirschfeld found among 648 orbital inflammations 408 that had their origin in the accessory sinuses.

The following two pictures show the way through which the inflammatory process spread to the connective tissue of the orbit in one case, to the optic nerve in the other.

The communication may take place directly through the hiatuses of the bony wall, subsequent to thrombophlebitis, lymphangitis and bone necrosis, or indirectly through the effect of toxins, as it is observed in cases of corneal ulcers which may be associated with hypopyon, in spite of an intact membrane of Descemet. Circulatory disturbances are also sufficient to produce a

defect of vision. At the beginning of this process the papillomacular set, which is known to be exceedingly vulnerable, suffers from the pressure due to perineuritis. In a later stage of the disease the interstitial neuritis descends to the optic nerve, thus directly affecting the papillomacular fibers. These anatomic changes throw light also on the pathogenic factors relevant to the so-called contralateral blindness. Suppurative sphenoethmoiditis of one side may affect the optic nerve of the other. This phenomenon may correspond with an anomaly presented by the communication between an asymmetric sphenoid or irregular ethmoidal cells of one side and the optic nerve of the other.

The suppurative character of this process is not imperative for such transmission of the disease. A mere inflammation of the mucosa may give rise to propagations. It often happens that the consecutive affection of the optic nerve survives the primary inflammation and even the recovery of the accessory sinuses. Paunz is fully justified in regarding those changes as analogous to the otogenic factor in the etiology of meningitis, facial paralysis and affections of the labyrinth. Similarly to ours, in those cases otitis media is the source of infection, and prompt intervention is the only means to prevent serious complications.

As shown in the foregoing, numerous anatomic evidences and pathologic arguments point to the important rôle of nasal disturbances in the etiology of retrobulbar neuritis. Nevertheless, there can be no doubt that, in addition to these etiological factors, multiple sclerosis proves to be a frequent cause of the disease. Their relationship is supported by the progress of the disease, as well as by indisputable pathohistological evidences.

These pictures demonstrate the histological changes referred to above.

For an effective comparison, I want to show you some slides of a normal dissected optic nerve, and others consecutive to tabes resp. neuritis.

Marburg found that from 10 to 15 per cent of his patients suffering from multiple sclerosis presented retrobulbar neuritises, while from 30 to 36 per cent of retrobulbar neuritises were the consequence of disseminated sclerosis. Sarbó records 350 cases of multiple sclerosis, 30 per cent of which presented pallor of the papilla and 29 per cent complained of failing vision. However, no special value can be attributed to these statistics as long as all

patients suffering from disseminated sclerosis are not subjected to precise eye examination.

Of course, patients suffering from multiple sclerosis may also develop retrobulbar neuritis secondary to nasal disturbances. Meller goes still further and insists that the changes consequent to sinusitis prove to be predisposing factors in the development of sclerotic foci. The recovery attained by means of operative treatment is no decisive factor in this question, as it is known that spontaneous bleedings, or a simple scarification may have a very favorable influence upon the disturbances of the optic nerve. Many more thorough examinations and careful and long investigations are indispensable in order to enable us to form a definite judgment as to the pathogenic and etiological factors. Trouble should be taken in every case of multiple sclerosis to examine the fundus of the eye, the eyesight, the visual field and scotoma, and in every case of retrobulbar neuritis neurological, endonasal and X-ray examinations are necessary.

In spite of all the difficulties, we are still able to draw important conclusions from a practical point of view:

In cases presenting positive rhinological findings, nasal treatment is always adequate. If conservative therapeutics, such as the application of wet compresses or adrenalin-cocain plugs, fail to bring relief within one or two weeks, there can be no hesitancy in advising surgery. The delay in surgical intervention is right on condition that rhinological findings are negative, also in cases in which neurological factors cannot be revealed either. But if there is no improvement observed and relapses occur, it is most advisable to open and clear out the ethmoidal cells and sometimes the sphenoid, in spite of negative rhinological findings.

We are aware of the fact that no operations are absolutely safe, but very often we are confronted with the apparent danger to which sight is exposed and there is a question of coming to its rescue.

We cannot be too conservative when dealing with cosmetic operations, but we equally cannot be too radical in indicating operations for the rescuing of vision.

The most important factors in the etiology of the chronic form of retrobulbar neuritis are the excess of the use of alcohol or nicotine. All signs give evidence that the damaging effect of nicotine surpasses that of alcohol. Traquair made a very extensive study of this form of the disease, and gave a report at the

meeting of the Oxford Ophthalmological Society, held this year. For that reason I confine myself to the statement that an early diagnosis and abstinence bring complete relief, but a delay in those leads to the development of partial atrophy.

The frequency of toxic retrobulbar neuritis presents a decreasing tendency after the great war. Retrobulbar neuritis associated with diabetes is beginning to be of very rare occurrence, since such remarkable progress has been achieved in the treatment of diabetes by means of insulin and dietetics. Occurrences of retrobulbar optic neuritis, consequent to lead poisoning, are equally diminished in number, as the progress in industrial hygiene offers effective means of protection against them.

Syphilis may be an indirect or direct cause of the disease. The optic nerve is sometimes affected by alterations or it is involved from troubles due to syphilitic lesions in the nasal sinuses. The favorable results obtained by antisyphilitic treatment are decisive as to this connection.

The hereditary form of retrobulbar neuritis deserves a special interest. It was first described by Leber in 1871. This disease is passed on by the female members of the family, but those subject to it are mostly men. (Gynaphoric type.) Vision usually begins to fail at the age of from 20 to 25 years; an absolute central scotoma is manifested very early. It never ends with total loss of vision, but does not recover either. Ophthalmoscopic examination reveals partial atrophy of the optic nerve, confined to the temporal margin of the papilla. The differential diagnosis often leads to special difficulties, but the course of the disease, as well as family history, help us to a final decision. The disease is of rare occurrence, but there is no doubt that this etiology in some cases never comes to light. Since the important rôle of hereditary agents has been discovered, the frequency of such cases is increasing. More attention should be given to such hereditary factors than has been before.

My grandfather called attention to those hereditary factors 100 years ago. His special interest was drawn in this direction by a family in which six children inherited the cataract of their father; all were operated upon by him on the same day. Nettleship, Fleischer and Clausen collected the material in Europe, and all clinicians of today enumerate a great number of family trees manifesting this heredity.

The following charts give a vivid picture of many such examples. The investigations carried out within the 60 years that have passed since Leber published his observations have reaffirmed his theories as to the character of the disease. Men are subject to this affection, but it is passed on by women. Women suffer from it in very exceptional instances, and it is more frequent in the descendants than among the generation of those who pass it on. Nettleship found among 360 patients men and women partaking according to the ratio 1 : 6 (the ratio in impaired color vision is 1 : 10). It is especially difficult to come to a correct diagnosis in these cases because altogether only a few members of the families are affected. The diagnosis is most difficult when facing the first case of the familial disease.

Zentmayer recorded valuable data from America.

I know of a very instructive occurrence in a family I used to know and still know. The first member had an attack at the age of 20 years. His family attributed his troubles to excess in the use of alcohol and tobacco, but it was soon revealed that he was a member of a great historic family in which numerous affections of the retrobulbar optic nerve had been observed in the past. Bishops, politicians and scientists were members of this distinguished family.

All the facts I have attempted to call your attention to point to the great importance of a thorough study of retrobulbar neuritis. Many questions have yet to be answered. The intense collaboration of laryngologists and ophthalmologists is already secured in the United States. Such a collaboration is indispensable if we want to solve the many problems still existing in this field.

DISEASES OF THE PARANASAL SINUSES AND THEIR RELATION TO DISORDERS OF THE EYE.

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An analysis of research study and clinical progress in otolaryngology during the past two decades shows a special interest in three great fields of work: (a) the laryngeal tract, so ably handled by Jackson and his school; (b) the ear and its intracranial complications, and (c) the paranasal sinuses and their focal pathology.

This order of arrangement conforms in a measure to the chronologic success in solution of the major problems involved in each. The investigation of the correlation of ocular and nasal pathology, though mature in record of research, still seems transitional and on the threshold of convincing scientific accuracy. The writer appreciates both the honor of sharing this discussion in such distinguished company and participating in a symposium on this closest link between the two specialties—almost the *raison d'etre* for the title of this great Academy.

The primary purpose in this paper, as designated by your secretary, was to review some of the more important theories in work along this line and to interpret, from an otolaryngological standpoint, the present status of this ocular and nasal interrelation. This brief study should perhaps be not so much concerned in differentiation and classification of diseases of the paranasal sinus group as in sinus disease *per se*, with less accent upon type of bacterial involvement than its virulence and possibility of active transmission. Certain disease entities in the accessory sinuses, such as tuberculosis, diphtheria, syphilis, malignant and semi-malignant involvements, have a definite ocular significance, but their symptomatology and laboratory diagnosis is a definite one, and their ocular as well as nasal management would assume specialized lines. The first problem of interest to this body is, What intranasal conditions, temporary or permanent, contribute to pathology in the optic tract, and how is this infection transmitted?

To obtain the composite of current opinion requires a somewhat careful review of the extensive literature upon the subject from the well laid foundations of Zuckerkandl,[1a] Onodi,[1b] Hajek,[2] Van der Hoeve,[3] Vossius,[3b] Loeb,[4] and others, with an emphasis upon anatomic conditions, pressure atrophy, dehiscence and necrosis of bony walls, to such later views as those of Sluder,[5] Skillern,[6] White,[7] Eagleton,[8] Gradle[9] and Wherry[10] in this country, St. Clair Thomson,[11] Logan Turner,[12] Maure[13] and others on the other side, analyzing simple nasal hyperplasia, toxic transmission, septic thrombophlebitis. This reveals how clearly the earlier writers foresaw these later developments of study, and it is only fair to give them prior credit for their insistence upon further light from microscopic and bacteriologic study of vascular and lymphatic transmission, to clarify their postmortem information.

Hajek,[2] in his work on the nasal accessory sinuses (1898), placed the order of frequency in sinus origin of orbital complications: Frontal, ethmoid, maxillary, sphenoid. Three predisposing factors were cited: (1) Thinness of the dividing walls, (2) dehiscence, (3) venous anastomoses. Under symptoms of inflammatory changes in the orbit, this author discussed orbital abscess and thrombosis of the central vein of the retina, describing symptoms in extension from frontal, maxillary and ethmoid invasion, and referred to Kuhnts'[14] three cases of thrombosis due to long standing chronic empyema of the maxillary antrum. Symptoms of disturbance from mechanical causes, included (1) displacement and disturbance of the mobility of the globe, (2) injury to the optic nerve, (3) changes in refraction of the globe, (4) disturbance in the drainage of tears. Finally, under symptoms of secondary inflammatory affections of the globe, were placed (1) affections of the conjunctiva, cornea and lachrymal apparatus; (2) affections of the uveal tract, (3) affections of the retina and optic nerve—all traceable to accessory sinus disease.

In the special diagnosis of the rhinologic origin of retrobulbar neuritis, Hajek gave four classes: (a) cases with pronounced disease near the optic nerve, (b) cases with hardly perceptible pathologic sinus change, (c) marked suppuration in the posterior ethmoid labyrinth and sphenoid sinus with bone destruction, (d) neoplasms in posterior sinus walls.

In the earlier group too much credit cannot be given to Onodi[1] for his splendidly illustrated study of topographical anatomy and

clinical observations, the German edition of whose work was published in 1906. He organized into twelve main groups thirty-eight varieties of morphologic relationships of the optic nerves, optic canals and optic sulcus with posterior ethmoidal cells and sphenoidal sinuses. Observations were made on the thickness of canal walls, dehiscence, congenital and other, and thinness of septa between adjacent sinuses; under clinical considerations he discussed mechanical and traumatic lesions producing pressure swellings, circulatory interference impairing optic nerve function, perforation of sinus walls from caries and necrosis, involvement of nerve in hyperostosis of the sphenoid wing, with narrowed optic foramen. The problem then as now was the lack of demonstration as to source, course and spread of infection by blood vessel route. After clarifying symptoms in orbital and retrobulbar area referable to the four sinus areas, he indicated his complete accord with prompt relief by nasal surgery but made these trite cautionary observations: First, that optic neuritis and optic atrophy may be accidentally associated with accessory sinus disease and not caused by it; second, operation does not always cure, and often there are spontaneous cures without operation.

Loeb and Wiener,[4] in 1920, presented an anatomic study of the sinus relations with clear cut illustration and based their investigations on these conclusions: (1) That lesions of the eye and adnexa followed nasal pathology much more frequently than ordinarily supposed, (2) that routine study of minor processes would be more fruitful than occasional striking exceptions, (3) that it is necessary to include the whole category of eye lesions in the study, and study all eye changes in the presence of every nasal disturbance. From such study much information is obtained—the changes in mucosa continuity of the lachrymal sac, pathologic evidences from dehiscence in bone, unusual variations in the relation of ethmoid and sphenoid sinuses to the optic nerve, deductions from vascular and lymphatic distribution, differentiation of influences upon various nerves distributed to eye service and analysis of direct results from malignant disease, tuberculosis, syphilis, diphtheria, as well as common pathogenic flora in the nose.

Beck,[15] in 1911, presented some fine studies of the pathologic histology in the middle turbinate, from ethmoid curettements, sinus wall linings and sinus contents in acute fulminant, chronic suppurative and chronic nonsuppurative types, demonstrating

changes in vascularity, with round cell infiltration, areas of edema, destruction of epithelium, pressure atrophy and, in acute types, actual osteitis.

Tunis,[16] whose anatomic work on the head by means of frozen sections has merited attention, followed in 1912 with a discussion of sphenoidal sinusitis in relation to optic neuritis. In five years' careful study he could find no peculiar distribution of lymphatic tissue between the sinuses and optic nerve, posterior ethmoid and sphenoid being equally intimate in relation to nerve, which would predispose to infection of the one by inflammation of the other. Continuity and proximity were the important factors. On the other hand, Coffin,[17] in 1917, discussed the interrelation of ophthalmic and ethmoid arteries and veins, in the effect upon ocular circulation of disease of the nasal accessory sinuses, and the likelihood of phlebitis of the ophthalmic vein from sinus empyema. He recalled a statement by Christian Holmes, ten years before, that 40 per cent of all affections of the eye, including asthenopic conditions, could be attributed to some form of sinus disease.

Vail,[18] in 1919, reports four cases of exophthalmos from abscess of sphenoid, posterior ethmoid, anterior ethmoid and frontal, respectively, in each of which an actual hole in the periosteum was found in orbital operation, and emphasized this view of septic transmission—but he also reported three cases of monocular retrobulbar neuritis from ethmoid disease, with initial negative rhinoscopic findings.

The late Dr. Skillern,[6] former president of this Academy, in his fine work on the nasal accessory sinuses, discussed thrombosis of the cavernous sinus and retrobulbar affections of the optic nerve, pointed out the anatomic position of the venous sinus against the lateral walls of the sphenoid cavity, ready infection through the bony walls and extension of pathologic process through lymph canaliculi or perforating veinlets. Barnhill, in discussion of orbital cellulitis and orbital abscess which sometimes follow suppuration in frontals, maxillary and ethmoids, concluded that pathogenic organisms enter orbital tissues through anastomosing veins, dehiscence in orbital wall or fistula necrosis. Eagleton's[8] view of thrombosis or, as termed by himself, septic phlebitis of large venous radicles, in its relation to orbital cellulitis and cavernous sinus thrombosis belongs in a measure to this field of discussion. Whether infection originated from lip, angle of mouth or lower margin of nose, or in acute involvement of

ethmoid sinus, there is infective phlebitis of the orbital veins. Favored by absence of valves in this area of transmitting veins, infection passes directly upward, without ordinary protective obstruction. This concurs with St. Clair Thomson's[11] penetration of sphenoidal boundaries, septic infiltration of venous walls, endophlebitis with coagulation of blood along the line of inflammation, clot accumulation until circulation is stagnant, fibrous degeneration of thrombus and, if infection be virulent, breaking down of clot into semipurulent mass, with parts thrown off at distal end of vein and thrombi caused in other veins. These views on thrombophlebitis might be considered part of the transitional stage in viewpoint.

Mention might be made of links in the infective chain aside from the spheno-ethmoid area. Dean[19] cites Turner's[12] belief in etiologic importance of the ethmoid in phlyctenular conjunctivitis and that improvement after tonsil and adenoid operation was due to their influence upon the ethmoid. Lemere[20] discussed ocular conditions dependent upon latent antrum disease, and believed ethmoiditis due to underlying antrum involvement of such low grade that classic antrum symptoms were often missing. Haskin[21] stated, in regard to the sympathetic nervous relation in the head, that the fibers of the carotid and cavernous plexuses are in intimate contact with these diseased sinus areas and cells. They are the vasomotor and trophic nerves, the pupil dilating nerves, the control nerves in certain eye and orbital musculature and belong to the sympathetic system. The pathologic relation is obvious.

Sluder[22] followed his earlier work in 1907 on blindness due to postethmoidal empyema, and in 1918 on headaches and eye disorders of nasal origin, by a somewhat exhaustive treatise, in 1927, upon nasal neurology, headache and eye disorders. This discussed at length hyperplastic sphenoiditis and its clinical relations to the neighboring ophthalmic nerve supply. He believed that optic nerve disorders were frequently due to hyperplastic bone changes in the spheno-postethmoidal district—narrowing the foramina. This might be a process of years or produce quick blindness, with or without changes in the disc. He suggested muscular imbalance from discomfort due to attachment to inflamed bone at the apex of the orbit.

In these progressive and more radical views, the pendulum may have swung too far, but the careful presentation of White,

with the help in pathology of Jonathan Wright,[23] would seem to indicate an honest effort either to substantiate older anatomic and pressure views or to deduce from them a more tenable field.

Gradle,[24] quoted by both White and Wherry, as early as 1915, reviewed the anatomic relations and blood supply of the optic nerve, presented by Loeb, Onodi, Vossius and Gray, analyzed the passage of infection or edema from sinus periosteum through diploic veins and lymph channels of orbital periosteum to intracanalicular portion of optic nerve dura or through periosteal or dural veins directly to central vein of Vossius, producing varied optic involvements, and concluded that antomic relations of sphenoid and ethmoid sinuses to optic nerve were immaterial—transmission was through soft tissues alone. White, in 1919 and 1921, presented his own careful studies on hyperplasia, even in the nose negative to examination and X-ray studies, and concluded that the dividing line between suppurative and nonsuppurative contributions to eye pathology was a difficult one; he believed noses filled with polyps and pus rarely produced effect on vision. White observed that up to a dozen years before these papers the intimate pressure relation of optic nerve to sinuses was stressed—now there was a growing comprehension of toxemia and hyperplasia. In relation to bacteremia of focal infection, he alluded to Billings'[25] blood vessel work—hematogenous focal infection, blood vessel and lymph borne, has been demonstrated in nervous apparatus—why not also in the optic nerve? In this conviction, though later more conservative, he removed the middle turbinate and opened the posterior ethmoid in all cases, usually the sphenoid, as well.

He, however, very fairly admitted that cases with multiple sclerosis, brain tumors and various constitutional diseases often have incidental nasal pathology, and toxemia may come from alcohol, tobacco, quinine, arsenic and lues as well as infected sinuses.

Your own secretary, Wherry, carefully reviewed the question of paranasal sinus disease and visual disturbances in 1925. He accepted Gradle's pathways of the optic nerve in the bony tunnel formed by posterior ethmoid and sphenoid, with its three divisions, (a) vascular, (b) avascular, (c) canalicular, with danger in the posterior canalicular portion where dura and periosteum are closely associated: edema of anterior portion of the nerve produces enlarged blind area and peripheral changes with good

prognosis; edema of canalicular portion would cause central scotoma and grave danger to vision. According to Wherry's view, venous and lymphatic drainage was a greater menace than actual contiguity of infected tissue. Localized hyperplasia was often more dangerous than protective pyogenic membrane.

The allergic side deserves a hearing in this discussion. In the question of operative indication or the reverse, we do not know how much bearing the allergic reaction, eosinophilia and vitamin deficiency background have upon the infective problem. Dean, Tobey,[26] Kern,[27] Coates,[28] Ersner[29] and others have expressed initial views and are working on the problem. It is not surprising to find many of the stronger specialists retreating to the more cautious position in the belief that systemic resistance and immunity still have a function.

This review has been rather arbitrarily selective, and much valuable reference has been omitted. Onodi alone, in his chapter on clinical considerations, gives some two hundred individual references. Our own literature is replete with similar observations. The writer would summarize the situation somewhat as follows: The paranasal sinuses are subject to infection from immediate intranasal conditions, and more often than suspected this is secondary to other contiguous foci, tonsils, teeth, etc. Such infection is favored by such anatomic blocking as the septum-ethmoid complex—a title rather arbitrarily selected for a presentation of this subject in 1924[30]—is probably increased by the intranasal results of allergic sensitivity, and is more or less due to incidental susceptibility to available infective organisms.

Such infection appears as a suppurative empyema of the sinus cavities, pathologic conditions of the sinus walls, with or without polypoid change—hyperplasia of the turbinate tissues, with frequent cystic degeneration—and may sometimes be present without obvious intranasal symptomatology or X-ray demonstration, though Law,[31] Pfahler,[32] Hirtz[33] (in France) and others have taught us much in the interpretation of sinus wall pathology and protective changes in surrounding bone. The crowding of deflected or irregular septum would seem to work in two ways—exerting pressure block, particularly in posterior ethmoid and sphenoid on side of convexity, and stimulating hyperplasia on the wider side of concavity.

In the present discussion, the question at issue is not sinus pathology *per se* but the existence of sinus pathology in known

disorders of the eye for which it may be the cause. Perhaps, as White expressed it, old necrotic walled sinuses with abundant polypoid granulations should freely produce eye complications and usually do not. Comparing frequency of sinus occurrence and times of actual eye involvement, this is true, but nevertheless this state represents a potential danger in the light of investigations on the thinness of sphenoid and posterior ethmoid walls and dehiscences.

In the search for obscure sinus etiology, the precision of ophthalmic diagnosis has its counterpart in the exhaustive intranasal search. Everything must be taken into account; external and internal nasal contour, character and location of intranasal discharge. Beside the help from X-ray and transillumination, under full shrinkage of tissues, and with nasopharyngoscope, the middle meatuses and neighborhood of the sphenoid ostia must be repeatedly asked to show the fine streaks of infective secretion which will be diagnostic. Perhaps culture of washings and lipiodol injections will be necessary for antral study. Hypertrophy, hyperplasia and vasomotor changes must be differentiated. If the infection is found, prompt surgery is indicated, for experience has shown how effective may be an inconsequential remaining tag of infected tonsil, a single carious tooth, or a limited sinus cell area as toxic origin. Fortunately intranasal surgery in skillful hands is comparatively safe. The inferior turbinate and septal mucous surface repair mutilation badly, with crusting squamous epithelium, but these seldom need be sacrificed. It has been the writer's experience that careful surgical exenteration of diseased ethmoid and sphenoid areas neither invites disaster nor leaves surface troubles equal to the original pathology.

One who is not an ophthalmologist may be excused for grouping the eye complications in three main classes: 1. Retrobulbar and optic nerve head disturbances; 2. involvements of the conjunctiva, uveal tract and disturbances of function, reflex in character and otherwise, and (3) orbital cellulitis and abscess, with and without exophthalmus. The first group would seem to justify immediate interference on any reasonable ground. As suggested in Peter's[34] presidential address in 1928, the sight of the eye is too important for any omission, with full agreement and understanding by the patient of possible result. The time element would seem to be of utmost consequence; even exploration of the posterior sinuses for ventilation or relief of pressure, as in case of intratympanic

pressure of the ear, might be of value. If necrosis is found, all operative doubt will fortunately be dispelled. In the third group, when the existence of orbital abscess is still in doubt and exophthalmos spells the danger of cavernous sinus thrombosis or meningitis, experience has justified prompt attack upon any suspicious sinus area. The major consequence of intracranial involvement is too great in comparison to any intranasal operative damage, and the results recorded have been sufficiently favorable. This too requires promptness and, in serious condition of the patient, sometimes courage.

Perhaps a personal viewpoint may be best given in conclusion by analysis of the two groups thrown upon the screen. These twenty cases were selected from the group of cases studied for their nasal and ocular pathology. The entire first group and the three cellulitis cases of the second called for immediate surgery. Four of the first group completely recovered, one without operation. Three registered some improvement, one marked improvement, and two none at all. All of this series had definite pathology, and the predominance of ethmoid involvement will be noted. A considerable number here and on the unreported list were returned "sphenoid negative" in the X-ray examination, and this was confirmed in large measure by local examination. You will recall Hajek placed the sphenoid last in order of frequency.

The cellulitis cases were reported in a previous paper[35] on the diagnosis problem in orbital cellulitis. Two recovered and one died, perhaps from too much, perhaps from too delayed interference.

The remaining seven cases of Group 2 included four recoveries, and definite improvement in two of the others.

It will be noted that this major group of cases included but one hyperplastic diagnosis. This scarcely represented the writer's view, for he is rather more nearly in accord with White's opinion, with this proviso that routine intranasal treatment should be given on adequate time interval. In some of these cases of recovery following operation, spontaneous recovery was still within possibility, though in one case of each of the above lists improvement was obtained only after repeated operative intervention in search for remnant focal areas.

The majority of otolaryngological surgery stands somewhat at mid-field, between the conservative and ultra positions. It is possible that this index point will move forward, though the more

intensive viewpoints will tend to recede. Even age lends to conservatism. Increasing general familiarity with the sphenoethmoid operative field will minimize its danger sequence, and the fascination of "angling in these hidden pools" is only equaled by the patient approach to the hidden recesses of the middle ear.

The older views of proximity of sinus walls to optic nerve and other ocular structures and the pressure etiology in infection should not be supplanted but reinforced by later understanding of toxemic transmission by blood and lymph channels, bacteremia from infective focus, the nature and sequence in thrombophlebitis and the operative indications in obstructive hyperplasia. The record of successful experience is so overwhelming that trustworthy ophthalmologic diagnosis with skillful and thorough sinus surgery would seem to have barely begun their great crusade in ocular salvage.

In last analysis, the problem of greatest interest to this body is, What intranasal conditions, temporary or permanent, contribute to pathologic manifestations in the optic tract, and how can they be removed? From the ocular standpoint, this involves utmost precision in diagnosis—from the rhinologic, after the diagnosis, avoidance of needless and completeness of justifiable, surgery.

GROUP 1—OCULAR AND NASAL PATHOLOGY

		Eye Condition	*Nasal Condition*	*Operative Work*	*Result*
1	A. R. B.	Hem. neuro-retinitis with thrombi (D)	Ethmoiditis, polyps (R), deflected septum	Ethmoid exenteration. Submucous resection	Marked improvement
2	E. S.	Sub-acute retrobulbar neuritis (L)	Ethmoiditis (L), infected tonsils	Removed tonsils. Left ethmoidectomy	Recovery
3	T. G. P.	Neuro-retinitis (R)	Ethmoiditis (R)	Anter. ethmoidectomy	Moderate improvement
4	G. D. L.	Acute retrobulbar neuritis (D)	Deflected septum, ethmoiditis (L)	Submucous resection. Anter. ethmoidectomy	No improvement
5	G. B.	Vascular spasms cerebral vessels (D)	Deflected septum L. ethmoiditis	*No operation*	*Recovery*
6	A. L.	Angio-sclerosis (D), macular degeneration	R. Antral disease	Antral drainage. Partial turbinectomy	Moderate improvement
7	M. E. M.	Acute retrobulbar neuritis (R)	R. ethmoiditis R. antrum	R. spheno-ethmoid. Antral drainage	Recovery
8	H. S. F.	Color scotoma (R)	Double ethmoiditis	Double ethmoidectomy	Recovery
9	F. J. O.	Tubercular choroiditis (D)	Double ethmoiditis	Ethmoid exenteration	No improvement
10	S. A. G.	Macular choroiditis (D)	R. ethmoiditis L. spheno-ethmoid	Double ethmoid and left sphenoid	Moderate improvement

GROUP 2—OCULAR AND NASAL PATHOLOGY

		Eye Condition	*Nasal Condition*	*Operative Work*	*Result*
1	L. E. D.	Acute choroiditis (D)	Septic tonsils. Double spheno-ethmoid disease	Removal tonsils (F). Double spheno-ethmoid	Improved after each. Relapsed
2	I. J. H.	Acute kerato-uveitis (D)	Right antrum. R. spheno-ethmoid disease	Antral drainage Right sphero-ethmoid	Recovery. Some opacity
3	M. B.	Anterior uveitis (R)	Ethmoiditis (R) Septic tonsils	Tonsillectomy	Recovery
4	A. P.	Choroiditis (R)	R. ethmoiditis	*No operation*	*Sl. improved*
5	M. F.	Acute plastic choroiditis (L)	L. ethmoiditis	Ethmoidectomy	Recovery
6	W. J. G.	Recurrent conjunctivitis (D)	R. ethmoiditis R. antrum	Ethmoidectomy. Antral drainage	Recovery
7	H. R.	Orbital cellulitis. Exophthalmos (L)	R. ethmoiditis R. antral disease	Ethmoidectomy.* Antral drainage. Submucous resection	Died—meningitis
8	D. H.	Orbital cellulitis. Exophthalmos (D)	Double ethmoiditis L. antrum disease	Ethmoid exenteration	Recovery
9	B. H.	Orbital cellulitis. Exophthalmos (R)	R. spheno-ethmoid disease	Spheno-ethmoid*	Recovery
10	O. N. R.	Infiltrating keratitis (R)	R. ethmoid hyperplasia	R. spheno-ethmoid	Improved

*Operated on by associate.

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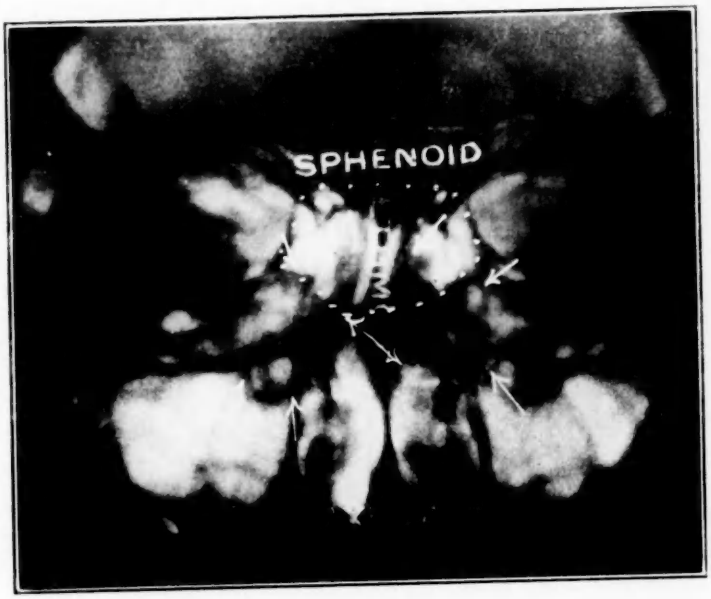

Fig. 1.

Fig. 2.

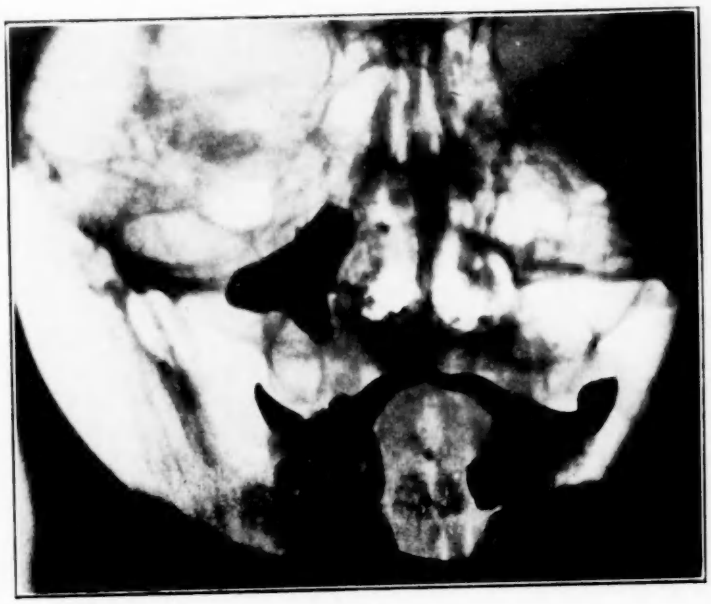

Fig. 3.

THE RELATION OF THE PARANASAL SINUSES TO OCULAR DISORDERS, ESPECIALLY RETROBULBAR NEURITIS.

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There are certain phases of the relationship between the eye and the paranasal sinuses which are apparently so well understood as to require little or no mention before a meeting of this kind. When one sees a case of frank orbital cellulitis now, I take it that the sinuses are always held as suspect and turned over to the proper authorities if nasal examination and roentgenograms offer evidence of infection. It has been my experience that where the sinuses are incriminated in such cases the infection is usually a purulent ethmoiditis or sphenoiditis, which leaves no doubt of its etiologic importance, and that in the other cases some cause for a metastatic infection can be located elsewhere in the body.

The sinuses as foci of infection causing uveitis, episcleritis and deep keratitis are also, I believe, kept sufficiently in mind by most ophthalmologists. The association is not nearly so frequent as one would expect from the frequency of sinusitis in our climate. Gilbert found 4 per cent of his cases of uveitis due to sinus disease. Bulson arrived at the same figure, while Brown and Irons found only 2 per cent, and Newton 8 per cent of such cases in their series of iritis. In a series of 118 cases of uveitis from our own office, 6.8 per cent seemed to be due to sinusitis.

The phase of this subject which interests us most, I believe, is the relative importance of the paranasal sinuses in the causation of retrobulbar neuritis. In spite of the floods of ink which have been spilled upon this question during the past ten years, it is still very decidedly a question. I think it may be resolved into three principal parts. In what percentage of cases of retrobulbar neuritis is sinusitis the cause? Can retrobulbar neuritis be caused by disease of the sinuses giving no evidence on rhinological or roentgenological examination, and, if so, what is the pathology

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of such sinus disease? In the presence of retrobulbar neuritis with no apparent cause and with no nasal evidence of sinusitis, shall we advise exploratory operation on the sinuses?

The first question has received widely varying answers. Herzog, of 28 cases, found definite sinusitis in four, while results of nasal treatment in twelve others led him to consider about half as due to sinus disease. Meller found 4.5 per cent, which he considered rhinogenic among the Vienna cases for two years. Lange found 3.5 per cent, Heine 3 to 6 per cent, and Langenbeck 3.5 per cent of such cases. Scheerer and Maier report on 203 cases from the Tübingen clinic seen during eight years. They found definite multiple sclerosis as the cause in 66 per cent, suspected multiple sclerosis in 5.5 per cent, tobacco-alcohol abuse in 5.5 per cent, and sinusitis in only 1.5 per cent. In 11 per cent no cause could be found.

Weill, out of thirty-five cases, found eighteen due to multiple sclerosis, two to tumors of the base, one to diffuse encephalitis and one to ependymitis. Only one showed ethmoiditis and in this case operation was of no effect. In his experience few cases have improved following sinus operations.

Redslob, with material from the same city, Strasbourg, is convinced of the importance of sinusitis in retrobulbar neuritis, as are other French observers, such as Worms and Rollet.

In America, following the work of Sluder and Wright, and the anatomical studies of Loeb and Schaeffer, a great deal of attention was paid to the sinuses. With Sluder's description of hyperplastic ethmoid and sphenoiditis, the rhinologist was no longer able to give a negative report when pus and polyps were not found in the nose, and the frequent association of the triological specialties with ophthalmology made a consideration of the sinuses a natural thing in cases of retrobulbar neuritis. It seems probable, also, that the large number of relatively young men with large private practices was partly responsible for our excitement over these cases; for the feeling that something radical must be done as soon as they were seen. A few dramatic results, such as often occurred, would offer nothing but support to such a course, while the possibility of a spontaneous recovery, such as was so often seen by Weill, Scheerer and others, was apt to be disregarded. It was very easy, under these circumstances, to fall into what Kalt has called a "therapeutic illusion." White states that his first seventeen cases were all considered

as due to sinusitis, while later experience has led him to consider the sinuses as only one and a relatively unimportant one, of the foci of infection to be considered. In sixty recent cases, he considered the teeth and tonsils to be responsible for 70 per cent, while only six showed ethmoiditis and most of these had other foci which were also considered important. The results of treatment directed to such foci were as good as those in his earlier series, 50 per cent regaining normal vision and 25 per cent being improved, while the others remained stationary.

While not many Americans have reported such large series of cases, Le Jeune, Bordley, Peter, Crowe and Oliver, Stark, Crane, Fitzgerald, and many others all reported cases in which the etiologic importance of sinusitis for optic neuritis was considered as proven, and the impression is gathered that most of these authors consider it one of the most important causes of the condition. Cushing goes to the opposite extreme, apparently doubting the rhinogenic origin of any of these cases. It is doubtless true that he has seen many patients with brain tumors who have recently undergone radical intranasal operations, and also that papilledema is a rare result of sinusitis. It is not, however, as he says, unbelievable that even papilledema could occur from sinusitis without increased intracranial pressure, since any inflammation involving pressure on the central vein in the subdural space could bring this about. Peter has reported elevation of the disc of three diopters, which subsided on opening the sinuses, and Ellett's case was similar.

Of thirteen cases seen by myself and associates during the past two years, multiple sclerosis was considered to account for three and purulent sinusitis for four. The remaining six cases must be considered undetermined. A nasal origin was considered probable in most of these, and all but one responded with improvement in vision to nasal operations or treatment, although in one nasal examination showed no pathology and in the others no definite sinusitis. Such a series shows how inconclusive any opinion based upon it would be without more exact records of nasal pathology than were to be found in our records.

Reasoning that if optic neuritis were frequently caused by sinusitis, a fair proportion of cases of sinusitis should develop retrobulbar neuritis, a number of observers examined cases of sinusitis for signs of nerve involvement, such as enlargement of the blind spot and central scotoma. Markbreiter found that

70 per cent of such a series showed field defects. Bordley found enlarged blind spots in 31 of 102 cases, Herrenschwand in 18 of 150 cases, Beck-Pillat in 6 of 53 cases of purulent sinusitis, 4 of 65 nasal anomalies producing poor ventilation, and in 8.5 per cent of 47 normal persons. Van der Hoeve found some changes in practically all cases of posterior sinusitis. Herzog examined 500 cases and found slight changes, chiefly enlarged blind spot, in 9.4 per cent, but definite signs which he considered significant of pathology in the nerve in only 2.6 per cent. In proportion to the number of cases, these definite signs were many times more frequent in acute than in chronic sinusitis. He concluded that while the slight field changes reported by some observers were probably not indicative of nerve involvement, such involvement does occur in a respectable number of cases without being noticed by the patient. While most of these defects disappear as the acute sinusitis clears up, some nerves remain permanently damaged, and this perhaps explains a certain number of cases in which optic atrophy is discovered later.

There are several reasons why these opinions as to the frequency of rhinogenic retrobulbar neuritis are so at variance, and why a review of them appears to leave nothing but confusion. One of these is that many observers consider recovery of vision after a nasal operation as definite proof that an infected sinus was the cause of the disease, while others, who have watched such cases clear up spontaneously, insist that many cases operated upon would have cleared up without this, and refuse to consider sinusitis as a cause without gross evidence of sinus disease. Another reason for confusion is the failure by one group of observers to have in mind the frequent course of multiple sclerosis, which often produces as its first symptom marked loss of vision. This may clear up completely, either with or without operation on the sinuses, and no other symptoms may develop for years; often not for ten years, according to Scheerer. I remember two cases which illustrate especially how easily these cases may be erroneously considered as due to sinusitis. Mr. J., a man, 31 years of age, came in with vision in the right eye reduced to 20/200, his first trouble dating from five days before. A central scotoma and contraction of the field was found and X-ray showed suspicious right sphenoid and ethmoids. These were drained, and vision twenty-five days later had returned to 20/20. Eleven months later it failed again to 20/70. Nasal ex-

amination was negative, but the spinal fluid contained 15 cells per cm., an increase of globulin, and gave a gold curve suggestive of organic disease of the central nervous system. Vision remained the same under treatment by a neurologist. A young woman with an exactly similar history had her ethmoids drained at the advice of my father, and regained normal vision. Five years later a neurologist in another city wrote that she was under treatment for definite symptoms of multiple sclerosis and that vision had failed considerably.

The opposite point of view, however, that of considering all cases of retrobulbar neuritis in which empyema of the sinuses cannot be demonstrated as due to multiple sclerosis, even in the absence of all other signs pointing to it, may prove to be equally erroneous. Thus it will be seen that the percentage of cases attributed to sinusitis in the various series quoted is very much a matter of the observer's personal opinion.

Which of these opinions we will accept will depend on our answer to the question, What is the pathology of sinus disease causing retrobulbar neuritis? Here we are met with a great deal of theorizing, and a few valuable studies, chiefly by rhinologists. White reported on the pathology of nine cases in which the tissue was examined by Wright. He found edema, fibrous hyperplasia and chronic inflammatory changes in the mucosa, which sometimes involved the bone. He considered this to be evidence in some cases of an inflammatory process involving the nerve by direct extension. Later experience caused him to change his opinion and to consider the tissue changes found too slight to support such a theory. In 1928 he stated: "I have never seen a case, except in malignant disease of the sphenoid, where I thought there was direct extension from the sinuses to the optic nerve." He believes the hematogenous route to be the almost exclusive method of infection. Hence he considers infections of the antrum as important for the nerve as those of the ethmoid which, in his later cases, he has rarely found. He considers a few cases due to hyperplasia of the turbinates, with obstruction to the ostia and a resulting negative pressure within the sinuses which favors absorption of toxins and bacteria from the blood stream. O. Beck, in sections of the sinus wall or turbinates of four cases found a catarrhal inflammation of the mucosa, which was infiltrated with eosinophils and plasma cells. There were no signs of acute or purulent inflammation, and he believes the changes should be

interpreted as the result of negative pressure in the sinuses producing a type of inflammation which may cause damage to the optic nerve. Bordley's experiments seem to me to offer valuable evidence in this same direction. He placed tampons over the sphenoidal ostium in eight cases of sphenoiditis with normal blind spots. Two cases showed field changes following this procedure, one developing a marked increase in the blind spot and a relative scotoma five hours later, which cleared up when the tampon was removed. J. Beck examined tissue in a number of cases and found a noninflammatory rarefaction of the bone. Oliver and Crowe report ten cases, with examination of tissue in all. While a few showed gross nasal pathology, in many no discharge or polypi were to be seen and X-rays were negative. These cases showed infiltration of the mucosa and submucosa with eosinophils and plasma cells. Bony changes were not described, and evidently the bone was not sectioned in most cases. These cases were all considered rhinogenic, after careful exclusion of other causes, and the authors were able in all cases to observe hyperemia surrounding the ostia of the sinuses with the nasopharyngoscope. They advise such examination before cocain or adrenalin has been used, as the use of these drugs conceals this important sign. Redslob showed sections of a child who had died of bronchopneumonia in which extension of a purulent sphenoiditis through the bone to the nerve could be seen. The few other postmortem observations similar to this were chiefly in cases of destructive purulent sphenoiditis. In Pickford's case a perforation through the sella allowed invasion of the nerve, with cocci demonstrable in its sheaths. Neither sphenoiditis nor optic neuritis were evident during life. Ten Doesschate reported sections showing extension from a purulent sphenoid to the nerve, which contained diplococci, in a case of fatal pneumonia. (de Kleijn and Gerlach perhaps refer to the same case.)

While these cases demonstrate the possibility of direct extension in severe purulent sinusitis, they seem less important for the pathogenesis of the usual case than another case of Ten Doesschate. In this case, also of pneumonia, acute rhinitis and enlargement of the blind spot had been demonstrated during life, and postmortem ten days later showed thrombosed veins near the cavernous sinus and edema of the optic nerve sheaths. Coffin observed at autopsy thrombosis of the veins in the ethmoidal wall, orbit and nerve. Through the kindness of Dr. MacDonald

of Toronto, I am able to show a section from autopsy in a case of pneumonia with no history of eye disease. This shows a definite sphenoiditis with infiltration and hyperemia of the cortical spaces in the bony wall, and an area of hemorrhage and infiltration separating the dural sheath of the nerve from the bone. The nerve itself shows no involvement.

If it were necessary, these cases offer definite proof that infection of the nerve sheaths can occur by direct extension through the bony wall of an infected sinus. I think it is admitted by even the most conservative that this does happen in occasional cases of purulent sinusitis. Herzog has tried to show that it may occur in a larger number of cases, some showing negative nasal and X-ray findings. He sectioned the middle turbinate and portions of the ethmoidal walls removed at operation in a number of such cases. He first studied the normal histology of the region and described the intimate relationship between the dural sheath of the nerve and the cortical spaces of the bone separating it from the ethmoid and sphenoid cells. Among twelve cadavers, he found all stages between a type with dense bone having few cortical spaces and a less common type with thin cancellous bone, whose cortical spaces offer free communication between the submucosa of the ethmoid and the nerve sheaths which form its periosteum and sends processes directly through these cortical spaces to the submucosa. He believes the rarity of this type accounts for the rarity of optic neuritis in comparison with the frequency of sinusitis. In the cases of optic neuritis which were relieved by nasal operations he found small celled infiltration of the submucosa, with many enlarged vessels, while the bone showed edematous cells with new bone formation and cortical spaces congested and filled with fibroblasts and lymphocytes, the picture of chronic productive osteitis. In several cases where nasal operation was not followed by improvement he found the sinus walls macroscopically and microscopically normal. (The following slides, made from Herzog's cuts, show the two types of normal bone and the tissue changes in a case of retrobulbar neuritis.) "It must be our duty," he states, "to explain the histopathology of rhinogenic disease in optic neuritis," a suggestion which I believe cannot be too strongly emphasized. His studies led him to believe in the direct extension of the inflammatory process to the nerve sheaths through the vessels and cortical spaces of the sinus walls. The effectiveness of his "Daueranämisierung" he

believed rests upon the reduction of this edema and inflammation in the bone and the hastening of the absorption which takes place spontaneously in most cases of acute sinusitis, as such treatment is known to do in acute rhinitis.

One of the few attempts to produce retrobulbar neuritis was that of Pichler. He injected dyes and bacteria into the antrum of rabbits and produced pansinusitis but no involvement of the nerve. Dyes stained the bone but not the nerve sheaths.

Meller, in his review of the subject in 1924, emphasized the importance of such pathology as Herzog has described, and quotes Neumann's opinion that the slight temporary nasal inflammations following coryza are more apt to produce optic neuritis than is chronic sinusitis with the production of polypi and other gross nasal changes. Hirsch describes his own histologic findings in five cases, in four of which changes much resembling those described by Herzog were observed. He considers that this with other histologic studies justifies the belief that nonpurulent, catarrhal sinusitis may cause retrobulbar neuritis, probably by direct extension, with inflammatory edema of the nerve sheaths.

These studies of Herzog, Hirsch, Oliver and Crowe and others do indeed offer valuable evidence that such a process may occur. How often does it actually occur, with relation to the frequency of retrobulbar neuritis? In other words, in how many of the cases where no definite etiology is found does it occur? This will not be known until a large number of cases have been studied more carefully, both histologically and clinically. In looking back, one is amazed at the amount of tissue from the noses of such doubtful cases which has been removed and simply relegated to the slop jar. Such tissue, if properly studied, may offer evidence of value, not only for science, but for the further treatment and prognosis of our particular cases. Negative findings will encourage further search for possible intracranial causes, or a prognosis having in view the possibility of future multiple sclerosis. A no less important part of such study is the closer cooperation of ophthalmologist and rhinologist, not only before, but after, any operation on the nose. The observation of Oliver and Crowe that hyperemia was to be seen around the ostia with the rhinopharyngoscope in all their cases suggests the great importance of such findings being made known to the ophthalmologist in every case, as well as a very careful report on the condition of

the mucosa at operation. The suggestion does not seem radical that in some doubtful cases resection of the tip of a middle turbinate for biopsy might afford information as a guide to further treatment.

What one's attitude shall be towards operation on sinuses which are only suspicious in the presence of retrobulbar neuritis will depend on his conclusions from these studies of pathogenesis. First of all, the other causes of such neuritis must be carefully excluded. It must be admitted that multiple sclerosis causes at least half of the cases and probably more than this. A careful neurological examination should be made in every case, since the finding of one other sign, such as the loss of abdominal reflexes, would offer very good evidence for a diagnosis of multiple sclerosis. This examination should include lumbar puncture, since, according to Stewart, 70 per cent of cases show a characteristic colloidal gold curve and 40 per cent an increased cell count. Acute myelitis, which is perhaps an acute form of multiple sclerosis, must also be considered, although it is not common. A boy of 16, apparently in good health, suddenly lost all light perception in one eye. The fundus was normal and neurological examination was negative. A week later, while attending a baseball game, he fell and was carried home with complete paralysis of the lower extremities. Under a course of foreign protein injections by a neurologist, he regained full use of his limbs and normal vision in three weeks. Dollfus has reported a case quite similar to this, and Ellett's case, with a fatal outcome from intercurrent sepsis, was of the same nature. Cushing has insisted on the frequency with which the optic nerve lesions resulting from brain tumors are taken for rhinogenic optic neuritis. Weill and Nordmann have reported several such cases in which diagnosis was particularly difficult. In one a unilateral central scotoma gave place after two months to bitemporal hemangesia, and X-ray confirmed the presence of a pituitary tumor. All of our modern means of diagnosis are none too complete for proper classification of these cases, lest time shall be lost in useless nasal surgery.

One of the great difficulties is to obtain all this information without loss of much valuable time. These cases should be regarded as emergencies, and the various examinations should be done at once as a matter of routine, without waiting for each successive report.

When multiple sclerosis and brain tumors are eliminated, as well as this is ever possible, also tobacco-alcohol amblyopia, and the small number of cases with definite purulent sinusitis, about 15 to 20 per cent of cases will remain with negative nasal and X-ray findings and no other apparent cause. In these cases waiting a week or longer would seem to be accepted by most authorities as proper, since it is during this time that many begin to improve spontaneously. This week need not be spent in waiting alone. Sweats and foreign protein injections have been credited with many good results, especially when multiple sclerosis is considered a possible cause. Since the reports of Herzog and von Herrenschwand the use of Daueranämizierung has been widely tried with results in many cases as brilliant as those of operation. Whether or not pathology is demonstrated in the nose, a trial of this method would seem indicated. White's use of 3 per cent ephedrin spray every two hours probably accomplishes the same result. Herzog's procedure consists in packing the region of the ethmoidal and sphenoidal ostia twice daily with gauze soaked in adrenalin, the pack being left for two hours. Von Herrenschwand has reported thirteen cases with good results obtained by this means alone, and Mylius and others confirm his reports. In three cases with symptoms of multiple sclerosis Herzog saw no effect of the treatment, and he uses the method in doubtful cases for diagnosis, believing that only rhinogenic cases respond to it. He quotes a case in which vision failed again twice after the treatments were stopped, to clear up permanently after the third course of treatment was instituted. Where such failure occurs after a primary improvement, he believes opening the sinuses or improving their ventilation by turbinectomy is indicated.

When no improvement occurs under general and local treatment, the time to wait before considering exploratory operation will vary according to the degree of visual defect. If the visual defect is great and blindness is threatened, I believe most ophthalmologists would advise operation after a week of observation; some would advise it sooner. White believes 50 per cent of cases recover spontaneously, 30 per cent require submucous resection or turbinectomy, while about 20 per cent require free opening of the ethmoid and sphenoid cells. I doubt if White's dictum that only cases with optic foramina smaller than the normal 5.35 mm. in diameter usually require operation has been very gener-

ally followed. In spite of his interesting observation that most cases with optic neuritis do have small foramina, if one's own nerve were threatened it is doubtful if he would allow a matter of 1 mm. in size of this canal to determine the procedure to be followed. The time limit during which some hope may be offered by operation is uncertain. Certainly as long as atrophy of the disc has not taken place, there is enough chance to justify operation where nasal pathology is suspected, and some cases with considerable atrophy have recovered good or even normal central vision.

SUMMARY.

1. In the causation of retrobulbar neuritis, multiple sclerosis is the most important disease, probably accounting for over 50 per cent of cases.

2. Cases due to purulent sinusitis comprise about 3.5 per cent.

3. Of the 15 or 20 per cent for which no cause can be found, opinions differ as to how many are caused by latent or hyperplastic sinusitis.

4. There is pathologic evidence that such forms of sinusitis may cause retrobulbar neuritis by direct extension.

5. Before resorting to sinus operations in such cases, every effort must be made to eliminate other causes, especially multiple sclerosis and brain tumor. Examination of the spinal fluid with reference to gold curve and cell count should be made in every case.

6. Tissue removed from the nose should be examined histologically in every case operated upon, since it is only through knowledge of the pathology in such cases that we may obtain a better understanding of the whole problem.

7. With our present information and methods of diagnosis there will still be a number of cases in which exploratory operation on the sinuses is indicated.

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OPHTHALMOLOGIC SESSIONS

SOME CONSIDERATIONS OF MUSCLE SURGERY: WITH SPECIAL REFERENCE TO THE TUCKING METHOD.

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The accurate measurement and correction of refractive errors, educative treatment to overcome amblyopia in very young children and orthoptic exercises for selected cases will result in full correction of functional strabismus and heterophoria in approximately 50 per cent of patients. The good results of stereoscopic fusion exercises in lateral heterophoria of lower degrees obviate surgery in very many patients. The excellent work now done by ophthalmologists in these directions is resulting in less need for surgical procedure and in more deliberate surgery.

The many individual technical variations of advancement, resection, tucking and recession operations offer sufficient testimony to the fact that the entire field of muscle surgery still remains a proving ground of methods. Every ophthalmic surgeon does not have sufficient material to acquire experience with many methods. Finding a procedure which is satisfactory or approximately so, he adheres to the operation which gives him the best results. A certain degree of eclecticism is necessary, and it is not possible to adhere to a fixed procedure in all cases. The ideal of precision would only be attained if one could apply exact measurement in each of the various procedures undertaken in muscle surgery. Since all muscles are not alike in length, breadth, thickness and insertion, and all nerve centers do not function similarly, this ideal cannot be attained. The correlation of millimeters of correction with degrees of deviation will probably always remain a more or less relative affair. This is particularly true in the correction of strabismus in young children, in whom it is impossible to obtain exact data regarding the relation of adduction to abduction or of convergence to accommodation. Each individual patient must be regarded as a separate problem. One should take into

consideration the effect of wearing the most careful and complete correction of the refractive error over a sufficient period of time, the "functional" correction of the existing muscular deviation thereby obtained, the duration of the deviation, the ocular excursions, any existing contractures, and particularly, as Duane has emphasized, the possibility of a basic vertical paresis. By these observations, together with cumulative experience, one finds that a relative amount of muscle shortening or recession, or both, will produce certain degrees of effect in end results in similar types of cases. Time and physiologic adjustment aid results enormously. Standardization of muscle surgery will only permit of the application of certain fixed principles under very similar conditions. Only mass experiences based upon collective data from many reliable sources will result in a consensus of opinion in evaluating certain generally approved methods of technic. Wide latitude of judgment will always prevail in dealing with the various factors involved in the surgical correction of heterophoria and strabismus.

The objectives to be attained, whether relief of asthenopic symptoms clearly due to muscular imbalance, functional restoration of binocular vision, or purely cosmetic correction of an unsightly deviation, are the chief considerations which enter into the surgical problem. For the first of these—that is, muscular asthenopia—it requires fine discrimination with careful study of systemic conditions, the general health, toxemia, nasal obstruction, the vocation and personal habits of the individual concerned, etc., to permit analysis of a given case. In young individuals with heterophoria, one may occasionally restore binocular single vision which was previously present only for distant or near vision, but not for both. This accomplishment may be secured if corresponding images result, and if orthoptic exercises are employed to maintain fusion. In older patients with heterophoria, operation should only be resorted to if binocular single vision of a high degree is proved and then only for the relief of symptoms. Binocular single vision is of much less importance than ability to use the eyes comfortably. Undoubtedly orthoptic exercises should be preceded by surgery more promptly in most convergence insufficiencies than is generally the case. We have found that tucking procedures, sometimes supplemented by partial tenotomy, are the safest and most accurate surgical aid we can offer heterophoric patients. On the other hand, too hasty operative correction of

muscular imbalance and actual deviation occasionally leads one into the pitfalls of muscle surgery, increased asthenopia or diplopia.

Patients with alternating strabismus present the least problem in muscle therapy. Their correction is essentially surgical. If approximate parallelism is safely obtained the patient is happy and his "entourage" is satisfied, unless the strabismus later becomes monocular. A few patients will develop binocular fixation even though the fusion remains defective. Tucking is particularly valuable for alternating strabismus in young children.

Correction of a monocular cosmetic defect, usually associated with amblyopia, is the commonest reason for surgical intervention. For these patients, the straightening of the deformity relieves self-consciousness and other psychic states which is often their reason for desiring operation. In children, whom nature aids in the matter of physiologic adjustment, and in whom the result is sometimes functional as well as cosmetic, early results may be more safely achieved by tucking than by other methods we have employed.

The authors realize the difficulties of presenting new ideas upon muscle surgery. Because of the increasing popularity of tucking operations we desire to record general experiences with our methods during the past nine years. It seems relevant, moreover, to *attempt* an evaluation of this procedure. Measurements of deviations, their relations to underlying refractive errors, records of the nature and extent of particular surgical corrections employed, and the end results, when obtainable, have been collected and analyzed. Because of changes in method and technic, the fact that measurements have been made by ourselves and associates in various ways—and on account of inability to follow up patients from a distance, it has been well nigh impossible to make a *precise* analysis. However, it is believed that personal observations and deductions are of definite value. Since 1920, the tucking procedure has been employed for 144 patients among a total of 197 muscle shortening operations. No advancements have been done, resection after the method of Reese being the only other shortening operation done. Our chief reason for favoring the tucking method was because of its simplicity and safety.

Muscle tucking is by no means a new procedure. Jackson credits de Wecker with first performing the folding or tucking opera-

tion. His purpose was to prevent change in the "plane of action" of the operated muscle. Knapp, in 1886, also proposed a method of folding which he described as "the same as Critchett's (advancement) without excision of a piece of tendon." Legleyze, in 1891, gave prominence to the method of tucking, and he may be properly regarded as the originator of this operation, modifications of which were developed by Savage, Colburn, Magnani, Suffa, Woodruff, Valk, Reber, Bannister and others. Maxwell, Clark, Green, George, Todd, Bishop and many others devised instruments to facilitate estimation or measurement of the muscle shortening and to simplify placement of sutures with greater exactness and ease. One of us, in 1921, designed a special instrument for clamping the fold of the muscle at its base and permitting introduction of sutures *beneath* the blades through the tendon stump. None of the previous instruments was designed for suturing in this manner. Although greater shortening may be obtained without the use of the tendon tucker, the use of this instrument permits the tuck to be held in perfect position, with exact placement of sutures in the muscle so that they may be tied without puckering. An instrument facilitates the tucking operation except when a very high correction is desired.

Tucking Technic.—The tucking operation now practiced by us differs from that described by one of us in two previous publications (Arch. of Ophth., July, 1922, and July, 1925): The modified tucker was notched at its center, and for several years two single sutures were tied outside the conjunctiva. Following the suggestion of Dr. Allan Greenwood, the blades now have a width of 9 mm. and are double notched. Three buried sutures are now used instead of the two previously tied outside the conjunctiva. Since silk has been used, the tucks are never prominent longer than three weeks, and the irritation and granulomatous humps experienced with catgut are absent.

The great majority of our operations have been in children under general anesthesia. A snug bathing cap is worn to keep hair out of the field. A towel strapped across nose and cheeks with sterile adhesive plaster prevents contamination from the anesthetic, which is administered with a mouth tube by means of the McKesson gas apparatus. Adults are given a hypodermic of morphin and scopolamin one-half hour before going to the operating room. Four per cent cocain is used as a local anesthetic, 10 per cent if there is unusual pain. Subconjunctival injections

are no longer used, as infiltration complicated the operation and is unnecessary. Infection has been entirely absent since employing thorough skin disinfection with *acetone mercurochrome, the use of rubber gloves, 2 per cent aqueous mercurochrome in the conjunctival sac before and bichloride ointment after the operation. All children wear a Fuchs' mask to prevent contamination from fingers during convalescence.

When a lateral rectus is to be shortened the conjunctival incision is made vertically well behind the insertion of the tendon, about midway between the tendon insertion and the canthus. When the inferior rectus is to be tucked, it is easier to pass sutures properly if the conjunctival incision is made directly over the tendon insertion. The conjunctiva is freed with the least possible

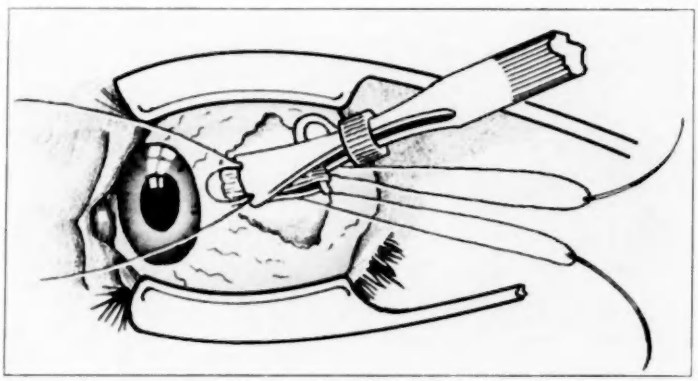


Fig. 1. Method of suturing with instrumental tucking.

disturbance of subconjunctival tissues. The tendon and muscle are prepared exactly as for Reese's resection, being exposed to their full extent with a minimum disturbance of the capsule.

The tucker hook, extended slightly beyond the open blades, is inserted beneath the tendon and drawn up, by means of a milled nut, approximately 1 mm. for each 2½ degrees of correction one desires to obtain. The tendon and muscle should be evenly adjusted so that the center of the tendon is over the center of the hook. Care is taken that the instrument is held vertical

*Note—Composition af acetone-mercurochrome solution.

Mercurochrome	40 gms.	Alcohol	1100 gms.
Water	700 gms.	Acetone	200 gms.

to the eyeball and muscle and not twisted sideways. When the desired amount of shortening has been obtained, the blades are pressed together and the ring clamp pushed downward gently, firmly, and as far as possible. It is important to be certain that the blades are snugly clamped before suturing.

Two quarter-curved, twelve millimeter needles are threaded on one number eight twisted white silk suture, twenty-four inches long. From the tendon side the needles are passed through the notches beneath the tucker. (Fig. 1.) The operator holds the tucker and grasps the needle as it emerges beneath it, while the assistant carefully keeps the capsular and conjunctival tissues from being drawn into the notch with the suture. The two needles are cut off, thus making three sutures. The border sutures are tied first. In using twisted silk one should tie each suture three times to prevent loosening before firm adhesion of the tuck occurs. The central suture is tied firmly with a surgeon's knot, reinforced with two single knots. If the central suture impairs

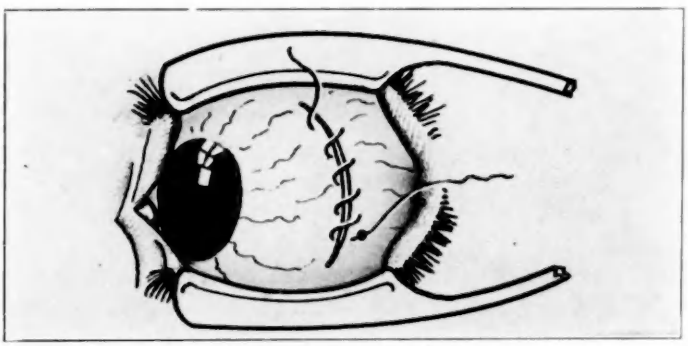


Fig. 2. The untied conjunctival suture.

the circulation it probably hastens atrophy of the tucked portion of the muscle. We have always tied the muscle across its entire breadth but have never observed any harm from it. All sutures are cut off close to the knots, remain buried, invisible and are absorbed.

Tension is now released by reversing the milled nut. The ring clamp is elevated and the tendon hook withdrawn. The fold of tendon and muscle is pushed backward evenly along the muscle but not sutured down, as it seems unnecessary.

The conjunctiva is closed by a running suture of number one twisted black silk with a knot two cm. from its lower end, which

is left exposed through the canthus. The last turn of the conjunctival suture is not tied after being drawn taut. (Fig. 2.) The suture exposed through the canthus may be removed by gentle traction on the fifth day. A child is usually unaware of the removal of this one external suture as it slips through the conjunctiva. No instruments are used, and the child has no fear or knowledge of "removing the stitches." Atropin bichlorid ointment is used in the conjunctival sacs, an eye pad and Blackman's two-inch elastic adhesive plaster is applied. This is not disturbed except for inspection and renewal of the dressing each day. Frequently both eyes are occluded.

Tenotomy of the central fibers of the opposing muscle and Curdy's recession suture (Fig. 3), as modified by Wilkinson, have been found valuable supplementary procedures. As a matter of fact, treatment of the opponent is always advisable when the deviation is marked and only one eye is operated upon. Very often one may prefer to postpone operation on an opposing muscle if

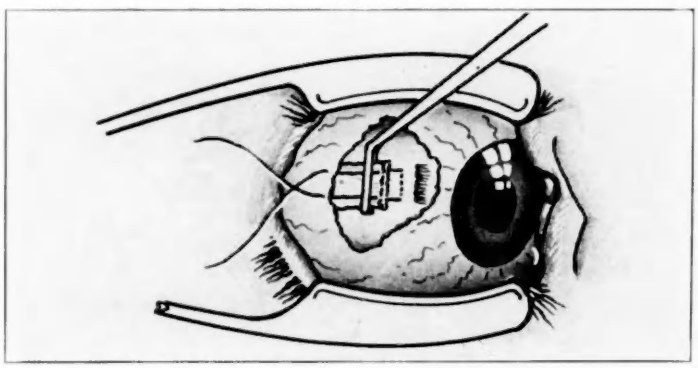


Fig. 3. The Curdy-Wilkinson recession

uncertain of the effect of the shortening obtained, but in certain high deviations it should precede it. Patients are informed in advance that one will probably accomplish a satisfactory result by one operation but that it may be necessary to do a second one later. This precautionary advice we have found very useful.

The most frequent question asked by observers of muscle tucking operations is, "How does one estimate the amount of tuck to be taken?" The answer to this difficult question is not easy. Judgment required in the correction of strabismus has rarely, ac-

cording to our observation, been based entirely upon actual measurement in millimeters. The old rule of one millimeter of shortening for each five degrees of deviation is by no means a reliable one to follow when resecting or tucking.

Comparing our experiences with those more recently published by Williamson Noble, Peters, Wilkinson and others, we have reviewed the results after resection or tucking operations have been performed. We have found that without tenotomy of the opposing muscle, the average correction in Reese operations has been approximately three degrees per millimeter of shortening. On the other hand, our figures in a series of tucking operations have been quite a remarkable revelation. We have found an average of two and one-half degrees per millimeter of shortening by

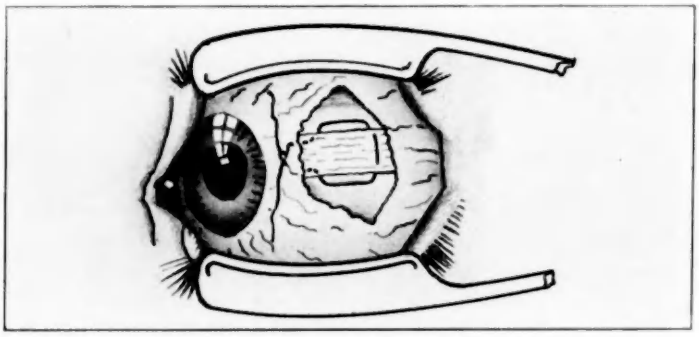


Fig. 4. Modified Legleyze muscle fold without instrument.

tucking operations to be more nearly correct when the opposing muscle is not operated upon.

One finds on exposure, various tendonous insertions, the capsule of Tenon and the muscle thickened or attenuated, and a tenseness or laxity of both. These anomalies can rarely be predetermined and influence judgment during operation as to the amount of tuck to be taken. The amount of correction which may be obtained by a reasonable and safe instrumental tucking of a rectus muscle, without tenotomy of an opponent, varies also with the particular muscle involved. For example, in tucking an inferior rectus for hyperphoria the shortening is relatively less than that required for an esophoria or exophoria. One may expect approximately three degrees for each millimeter of folded muscle.

In a case of exophoria of twelve degrees with adduction only slightly less than abduction (e. g., adduction ten degrees), one may expect only about two degrees of correction for each millimeter of folded muscle. Likewise, an esophoria of twelve degrees with high adducting and low abducting power will also require a tuck in the externus of about one millimeter for each two degrees of imbalance. Two millimeters of fold of muscle is the minimal amount which may be clamped and sutured. From these illustrative examples it will be readily and correctly inferred that for low degrees of heterophoria, when the tendonous portion of the muscle only is tucked or folded, a relatively smaller correction is obtained. It is in heterophorias, and particularly exophorias, that instrumental tucking procedures permit finer adjustments and corrections. Landolt firmly advocated operation

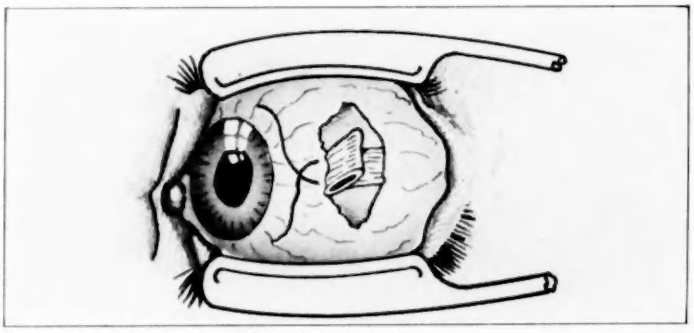


Fig. 5. Silk suture tied outside the conjunctiva.

on both eyes unless one is dealing with marked strabismus involving an amblyopic eye. This rule applies particularly to the correction of heterophorias and alternating deviations.

In convergent squint, seven millimeters represents about the actual amount of tucking which an external rectus muscle will safely stand and still permit easy insertion of the needles under the notches of the tucker. The same amount of correction per millimeter is obtainable in the case of the internal rectus, but this muscle will permit of only about five millimeters of easy instrumental tucking and passing of sutures. The following table of approximately maximum corrections is compiled from our recorded experience in tucking individual muscles *without tenotomy* of the opposing muscle.

Inferior rectus, maximum correction obtained.. 12°
Internal rectus, maximum correction obtained.. 12°
External rectus, maximum correction obtained.. 16°

When it is desired to secure a greater correction than can be obtained with a tucking instrument, we have found that twenty degrees may be secured on either the internal or external rectus by a modified Legleyze method, with silk sutures tied outside the conjunctiva. The objection to this procedure is the puckering of the muscle which results. It is not desirable to attempt higher than a twenty degree correction on a single muscle. Higher degrees of correction may always be obtained by operation upon the opposing muscle. If tenotomy of all central fibers of an opposing muscle is done, leaving only the extreme lateral attachments, the effect of tucking is augmented by about five degrees. In our series no complete tenotomies of the vertical or internal recti have been done. In several instances in which central tenotomy of the externus has accompanied tucking of an internus, or was done later, the effect of tucking was not increased more than five degrees. Experience was the same when tenotomy accompanied resection after the method of Reese.

Recession has been found to be a valuable aid in muscle tucking. R. J. Curdy, in 1916, proposed recession of the opponent with the use of the scleral suture to accompany advancement. He advocated equal advancement and recession—that is, if one muscle was advanced three millimeters, the other should be recessed by an equal amount. This principle, as recently explained by Rönne, is proving quite correct in practice, especially when dealing with a monocular strabismus of high degree and if the operation is confined to one eye. When the method of Curdy, as modified by Wilkinson (Fig. 3), has been employed, one millimeter of recession is equal to almost four degrees of correction, but results are not uniformly accurate. If recession precedes the tucking operation the effect of the latter is considerably increased. In all cases of monocular strabismus of high degree, when one desires the highest possible correction with operation on one eye alone, recession of the opposing muscle should precede tucking. By recessing the opponent almost to the equator and tucking the external rectus to the utmost degree, it has been found possible to correct a convergent strabismus of thirty-five degrees. It has not been possible to equal this degree of correction by reversing

this procedure with a divergent strabismus, and it is not advisable to attempt correction of over thirty degrees of divergent deviation by a combined operation upon one eye alone.

In alternating strabismus, the tucking should always be done equally upon the two eyes. Thirty degrees of deviation has been corrected in alternating convergent strabismus by tucking both external recti without tenotomy of the interni. In such cases, when tucking both external recti, the opponents have not been tenotomized or recessed at the same time, and if required, this has been done at a later period. In alternating convergent deviations, application of the Curdy-Rönne principle, above mentioned, in actual practice sometimes results in overcorrection. On the contrary, in divergent deviations no double tuckings of the internal recti have been done without tenotomy or recession of the externi.

In general, the following examples show some approximate procedures for various muscular imbalances:

Exophoria—12° distance, 20° near

Tuck each internal rectus 3 mm. or
Tuck one internal rectus 3 mm. Recess external rectus 2 mm.

Esophoria—12° distance, 20° near

Tuck each external rectus 3 mm. Leave the interni alone.

Hyperphoria 12°

Tuck one inferior rectus 4 mm. or
Recess one superior or one inferior rectus 3 mm.
Instrumental tucking of the superior rectus is not practical.

Alternating Convergent Strabismus

20°—Tuck each external rectus 5 mm.
30°—Tuck each external rectus 7 mm.
40°—Tuck each external rectus 7 mm. and
recess internal recti later when necessary.

Monocular Convergent Strabismus

20°—Tuck external rectus 4 mm. Recess internal opponent 3 mm.
30°—Tuck external rectus 6 mm. Recess internal rectus 3-4 mm.
40°—Tuck external rectus as much as possible. Recess internal, 4-5 mm. Operation on other eye later if necessary.

Alternating Divergent Strabismus

20°—Tuck each internal rectus ad maximum. Recess later if necessary.
30°—Tuck each internal rectus ad maximum. Recess each external rectus 3 mm.

Monocular Divergent Strabismus

20°—Tuck internal rectus 5 mm. and recess external rectus 4 mm.
30°—Tuck internal rectus as much as possible and recess external rectus almost to equator.

Paralytic Strabismus

Tucking is not a practical procedure in paralysis. Resection of as much of the flaccid muscle as possible, combined with recession, gives the best results.

Chief of the advantages which recommend the tucking operation is its safety and ease of performance. This makes it a valuable procedure for the surgeon who does not operate daily. It seems unnecessary to argue for preservation of the original insertion whenever this is possible, but it is a real advantage, obtained only by this method of shortening. Patients are occasionally seen after a tenectomy or an advancement in whom definite cyclophoria or vertical deviation has resulted in the operated eye from faulty insertion, maladjustment, cutting out or slipping of the sutures. Silk sutures securely tied in the tendon stump do not easily become loosened or cut out, and the correction obtained when the operation is completed is more permanent than by any other method. No suture troubles have been observed by us after tucking with the method now used.

The disagreeable hump previously encountered is no longer an objection. With buried silk, and by turning the muscle and tendon backward, the tucked tendon soon atrophies. Prominence is rarely noticed after the second week. Moreover, the ability to avoid removal of sutures which often requires a second anesthetic in the case of children is a decided advantage.

Practically the only argument against this procedure has been the inability of the operator to estimate the amount of tendon stretching. If the figures given in our estimates are followed the amount of correction obtained is very satisfactory, and if a proper allowance is made for stretching, the results are what they are planned to be. One may obtain almost as high a correction as by resection and one need not fear overcorrection unless the opposing muscle is tenotomized too freely or the recession suture is omitted. Errors in judgment are usually on the side of safety.

We believe that the tucking procedure is the method of choice for all heterophorias where finer corrections are necessary in overcoming insufficient action of certain paired or single muscles. In all cases in young children, not only because of its safety but because surgical correction is possible at an earlier age, when there is greater probability of obtaining binocular fixation and sometimes good fusion power, tucking is an advantageous operation. This is especially true in alternating squints, which sometimes later become monocular deviations. In these cases the operation should be done on both eyes equally or nearly so. It is a desirable operation in monocular strabismus not exceeding 20°.

For deviations higher than 20°, tucking should usually be combined with a recession. If sufficient correction is not obtained by both operations upon one eye alone, the other eye should have a supplementary correction at a later period. The use of some form of instrument is advisable, and after trying out nearly all the types designed the present model, manufactured by Victor Mueller & Co., of Chicago, has proved most satisfactory.

This paper will be illustrated by moving pictures of actual tucking operations.

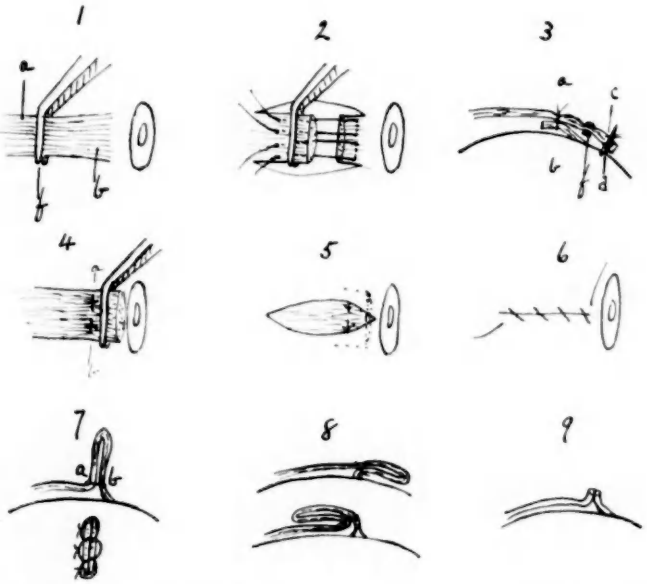
DISCUSSION.

DR. W. B. LANCASTER, Boston: There are several very good methods of shortening the distance between the origin and the insertion of a rectus muscle. Van der Hoeve lists fourteen types with seventy-five or more ways of doing it. I think we can limit the successful, desirable methods to a very small number.

There were one or two questions raised by the paper which I will briefly discuss. The problem is this—to move A over to B, Fig. I, thus shortening the distance from the origin to the insertion. The old method of advancement was to move c to d, making a new insertion near the cornea, which was supposed to have the advantage over the old insertion of giving a greater contact arc. We know now that what we thought were the advantages of advancement do not exist and that the muscle becomes reattached at the old insertion. To shorten the distance between origin and insertion there are really two ways: you can cut out a piece of the tendon and muscle and move the point forward, or, instead of cutting out a piece you can fold it in some manner. There are several ways of folding the muscle. This figure represents the fold; a is moved up to b. If you follow the English, a maritime people, you will call it a "reef"; if your interests are more feminine you will call it a "tuck"; if you are interested in horses you will call it a "cinch." But they all designate some method of folding the muscle, and they are all good methods. You can get a good result by any one of these methods, either tucking, reefing, cinching, or by resecting. The question then is whether you can make the shortening more *accurately* by a tuck or by resection. For some surgeons one method will be the best, for others a different method.

The method that I devised many years ago and described before this Academy in 1915 or 1916, was an advancement; and only those who practiced thirty years ago realize what a bias the profession had in favor of advancement. Snellen devised a very good resection operation back in 1869, but himself abandoned it for an inferior advancement method. (See van der Hoeve's monograph in Graefe Saemisch Hess, 3rd edition.)

My advancement made a new attachment near the cornea at d, Fig. II. I put in a stitch at d, and then, to favor reattachment at the old insertion, another stitch at b called a security stitch. I soon found that a good firm hold at b made the stitches at d superfluous, so I put in two stitches at b



Figs. 1-9. *a* point to be moved forward to new attachment; *b* new attachment at old insertion; *f* advancement forceps applied just in front of point to be advanced to new attachment. This can be measured with great accuracy because the muscle is not stretched before the point is selected.

In old style advancement *a* was moved to *b* by advancing *c* to *d*. Fig. 3 at *c-d* advancement suture, at *a-b* security stitch devised independently by Ewing, Lancaster, Duverger and others. The advancement stitch was abandoned and two stitches put through the old insertion, Figs. 2 and 4.

Fig. 5 shows the opening in the conjunctiva through which the work was done, now ready to be sutured as in Fig. 6.

Fig. 7. Tuck or reef showing also the triple suture of Burch which ligatures the whole muscle and may be followed by a slough, as Fig. 9. This does not occur with the overlapping suture *a-b*, Fig. 3.

Fig. 8. Two ways of disposing of the tuck or reef.

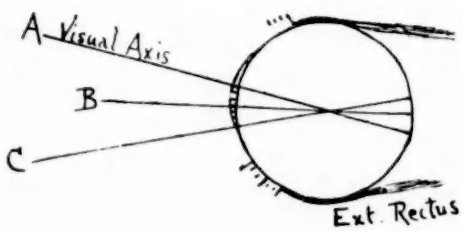


Fig. 10. To move *A* 3 mm. to *B*, the external rectus must be advanced or shortened 6 mm. This will result in moving forward the insertions of both internal and external recti 3 mm., dividing the effect between the two muscles and increasing the tonic pull of both, since they would not balance each other if one were under greater tension than the other.

and left out the advancement stitches near the cornea at d, Fig. IV. In other words, a simple resection. I claim that putting these stitches through the tendon stump at b, Fig. I A, and out through the muscle at a, pulling the muscle forward and tying a over b, is a more accurate method of measurement of the amount to be shortened than by the tuck. In case of the tuck you are stretching the muscle, and stretching it more and more the farther you screw up the tucker, and you have to know from experience how much to allow for stretching. In case of the resection you simply mark the point where you will put your forceps before the muscle is disturbed at all except by separating it from the capsule of Tenon.

In the tucking operation as done by Dr. Burch, when the three sutures are tied the upper fold is completely ligatured and sometimes sloughs off, and you have a hold which is, I believe, less secure than the resection. Therefore, since the tuck seems to me less accurate than the resection and doubtfully more secure, I seldom do it but generally do a resection.

The author said something about how to estimate the amount needed. Suppose this is the eyeball and here is (Fig. V) the visual axis (A); you wish to move it over to C, 6 mm. You advance the external rectus 6 mm., and what happens? Before you do the advancing these two muscles are balanced with a tonic pull of say 5; when you move the muscle forward 6 mm. you have increased the pull of this muscle by six units. It now is under a tension of say 11, and its opponent under only 5. As the eye turns into its new position the external rectus pulls less and less, and the internal more and more. When the external rectus has moved back 3 points so that it pulls 8, the internal rectus has been pulled forward 3 points so that its tension is 8, and they balance again in the new position. Thus if the opposing muscles are of equal elasticity or "strength," moving one 6 mm. forward results in a final shift of the eyeball 3 mm. toward that muscle, and 3 mm. away from its opponent. The optic axis is moved 3 mm., and both muscles are under an increased tension, tending to pull the eye back into the orbit.

But you cannot solve the problem in that easy manner. You cannot say if you shorten this muscle 6 mm. that you are moving the eye 3 mm. That depends on whether these two are equal. If one muscle is stronger, thicker, and harder to stretch, moving it 6 mm. will pull its opponent more than half way, and the net result is not 3 mm., but perhaps 4 mm. On the other hand, if it is more slender and more easily stretched than its opponent, the net result will be less than 3 mm., perhaps 2 mm., or even less. Moreover, in a large degree of convergence other muscles beside the external rectus must be taken into account. In this position the superior and inferior recti act powerfully as adductors aiding the internal rectus and adding to the amount that the external rectus has to balance. So you must allow a good deal more than one-half in deciding how much gain you will obtain. Thus each case is a special problem and cannot be solved by rule of thumb. Rules and averages are very helpful, but experience must be relied on for the final decision.

Dr. Luther C. Peter, Philadelphia: The role of tucking in ocular muscle surgery is gradually assuming definite indications in definite conditions. When well performed it has specific value, is easily executed, and

the convalescent period is somewhat abbreviated as compared with some of the other methods. It has its disadvantags and its limitations.

In the matter of technique, exception may be taken to the central suture employed by the authors of the paper. They, fortunately, have not observed any untoward results in these cases in the matter of necrosis. Some few years ago a series of five cases was reported by Dr. Goldberg before the College of Physicians of Philadelphia, in each of which necrosis developed and the results were most unfortunate. In these cases a double suture was used from the center, above and below. Nutrition was shut off, with the unfortunate result mentioned above. In my experience the central part of the tendon should be left intact. If two sutures are properly placed above and below the center, the end results will be satisfactory and necrosis need not be feared even by the tyro in surgery.

The essayists have referred to the disadvantages, which are not serious. The use of buried silk sutures in their hands solves the problem of obviating the tumefaction which often persists for considerable periods but finally disappears. If ten-day chromic catgut is selected, instead of the usual twenty-day material, the results are equally gratifying and I believe it is better suture material than silk.

One phase of the end results which was not discussed, but which I have observed, is the smoother appearance of the conjunctival wound after resections and advancements as compared with tucking. Some thickening may remain after either operation, but in my experience it is less marked after an advancement or resection than after a tucking.

As to its application and its limitations, I can agree entirely with the essayists in its ideal adaptation to the correction or relief of the phorias. It is equally happy in relieving a convergent squint of fifteen degrees or less, or a divergent squint of ten degrees. When applied to the external rectus more than fifteen degrees of correction can be obtained, but it is inadvisable. When a greater gain than fifteen degrees is desirable a supplementary recession of the internal rectus to its proper place on the sclera will yield better results without the danger of enophthalmos.

The authors of the paper referred but briefly to a partial tenotomy of the internal rectus. If there is an indication for a partial tenotomy it might be used when too much is attempted by a shortening of the external rectus, to relieve the tension on advancement sutures, but even then all but the most peripheral fibres above and below must be severed. The technique under such conditions is quite as formidable as that of a recession operation which yields definite values.

As has been pointed out, it is most difficult to tell the beginner how much muscle to excise, how large a tuck to make, or how much to recede the internal rectus to give certain results. Each case is a law unto itself and experience alone can guide the surgeon. Attempts have been made to provide scales in millimeters to guide one in accomplishing certain results. Such guides may help the beginner in his first cases, but as his experience grows he will find so many factors enter into the calculation that a rule of thumb must eventually give way to good judgment. The proper procedure is to operate on an external rectus and if the residual squint requires further operative procedure for its correction, a recession of the correspond-

ing internal rectus may be practiced, or at a later day the other external rectus can be shortened. In a general way the ratio of the recession to the tuck is approximately one to three in millimeters. Naturally this will vary with the size of the muscles, their attachments, the age of the patient, and the degree of squint to be corrected.

If the ease of performing a tucking operation, with its comparatively short and uncomplicated convalescence, will induce surgeons to operate earlier on children than is the usual custom, the operation is to be commended. In my experience, however, it is not the operation of choice which can be practical under all conditions, but should be selected as a corrective measure with due regard for its advantages, its limitations, and its indications.

Dr. S. B. Muncaster, Washington, D. C.: The best results that I have had in muscle operations have been from tucking the muscle. There are muscles attached so far back on the eyeball that one cannot tuck, and also atrophic muscles that will give but little result if tucked. I will illustrate this by citing two cases of operation for exophoria. One had 18° and the other about 33°. In the first patient, a woman who had good vision in each eye and 18° of exophoria, after dissecting the conjunctiva and Tenon's capsule the entire muscle was cut. A Maddox rod was used and the streak of red light passed through the candle. I thought it not advisable to advance the internal rectus. Six months after she returned with an exophoria of 11°. The conjunctiva was dissected up and Tenon's capsule dissected one-quarter around the eyeball; all the muscle was cut again. The muscle attached itself back of the first operation; with Maddox rod the red light passed through the candle. A test was made eight months after and the red light still passed through the candle. When the operation was performed I believe if a tuck had been made on the internal rectus the whole operation could have been made at the same time.

The second case was of a young man aged 21 years, who wanted an operation on an amblyopic eye for cosmetic effect. He had an exophoria of 33° The internal rectus had been operated on when the patient was a child. The conjunctiva and Tenon's capsule were dissected and about 5 mm. cut off the muscle. The eye looked fairly straight. The young man left for Florida, and when he returned in one year he had about 14° of exophoria. A second operation was performed by dissecting the conjunctiva from Tenon's capsule half way around the eyeball, and the muscle cut again. After two years he now has only about 4° of exophoria.

What I want to prove is that by the second operation for exophoria, where the muscle attaches back of the first operation, one can get as much as 10° more correction. This second operation will help when you want to correct a high degree and only want to operate on one eye.

Dr. Roderic O'Connor, San Francisco: I wish to compliment Dr. Burch on his very complete consideration of the subject as he sees it. I hope he will not feel hurt at my discussing it as I see it.

My shortening method has evidently escaped his attention, else why state that the tuck is the only one that does not disturb the insertion. The mere idea of "receding an internus to the equator" makes chills run up my spine. I have seen a total absence of inward rotation due to a set back of

4 mm. I wonder what he means by "making an allowance for tendon stretching?" He must have inadvertently overlooked the anatomical fact that tendon tissue is inelastic—necessarily so.

For many years I have felt that our troubles in surgery of the ocular muscles are due to habit contractions and relaxations (more especially the latter), as explained by Sherrington's Law of Reciprocal Innervation. Shortening a muscle does not abolish its habit impulse to relax which permits the eye to return more or less to its original position. I have seen a 25 (arc) degree of overcorrection, maintained for four days behind cover, disappear over night after uncovering the eyes. No change could have occurred at the point of shortening because the shortening material was still in place. Habit relaxation is, in my opinion, the only explanation.

The Doctor's system of handling hyperphoria can be explained only by the fact that his tucker cannot, as he admits, be used on a superior rectus, which is nearly always the paretic muscle and therefore the one to be shortened. If this idea is correct, and I am sure it is, shortening a left inferior rectus to correct the results of a right superior rectus paresis is as reasonable as shortening a left externus to correct an exophoria due to a right externus paresis. Shortening a normal muscle (the opposite inferior rectus) and recessing two normal muscles (inferior rectus of the same side and superior rectus of the other) simply because the tucker cannot be used on the muscle at fault is, in my opinion, wrong. Moreover, recession of an inferior rectus might result in diplopia on looking down. Having shortened the vertical recti in approximately 150 cases I feel somewhat qualified to make positive statements. In not more than six of these did diplopia tests point to an inferior rectus as the paretic muscle. I wish to repeat that an accurate diagnosis of heterophoria in kind or degree cannot be made without the aid of prolonged monocular occlusion. Those who refuse to use this important diagnostic aid are more ignorant of actual conditions than they are willing to admit.

As to judging results, degrees of elevation mean nothing and I stopped making such measurements many years ago. In the absence of tenotomies I find it almost as difficult to correct a 10-degree exophoria as a 40-degree squint. This because of muscle action under Sherrington's law. One has only to remember that patients' eyes will often be perfectly straight with their glasses, but deviate ad maximum without. A reasonable shortening often gives a full result; if not, its mate of the other eye must be operated on. Excessive shortenings may interfere with parallel action of the eyes, especially when combined with tenotomy of the opponent.

As to tenotomies, my scheme is what I call a two-stage tenotomy. The first is a central cut leaving a 2 mm. margin. Sometimes this is enough to break the habit spasm; if not, about six weeks later the margins are cut, but avoiding the central portion which has become united to the sclera slightly back of the original insertion. The final effect is that of a slight recession, but without at any time losing control of the globe and without the use of scleral sutures which are always risky, and especially so in the thin sclera of a young child.

The operation should be done as soon as nonoperative measures have failed. I have never seen glasses that were a full and accurate correction

straighten the eyes, unless they did so at once. I have frequently seen eyes straighten at once on giving such a correction in place of a partial and inaccurate one. So, in my opinion, if a second refraction after some months is markedly different, the first was, in all probability, wrong.

I like to operate early in order to increase the patient's chance of learning binocular vision in the normal way, this because orthoptic training is impracticable in small children and because but few mothers will persist in the prolonged and tedious alternate monocular occlusion necessary to save the sight in each eye.

Dr. William Thornwall Davis, Washington, D. C.: May I ask Dr. Burch just what is meant by full correction of functional strabismus, as mentioned in the first paragraph? Does he mean that the patient has correction of the squint with or without glasses? Is the squint considered cured if it is necessary for the patient to wear glasses for its correction? How long does Dr. Burch continue the treatment with glasses, patching and orthoptic exercises, before operating?

The evil result of the squint and the wearing of glasses would seem to make it desirable to obtain a cure before the school age, by surgical procedure, if this be necessary. One finds many mothers and children who do not cooperate in this difficult procedure. Hence the progress of these cases is often unsatisfactory. Again, the wearing of the full hypermetropic correction over the developmental period of life often makes it necessary for this individual always to wear glasses. Even though the squint is cured, the accommodation is so affected that the patient cannot see clearly or comfortably for distance or near without glasses. Hence the advisability of early operation and early cure of the squint without glasses.

The tucking method as shown by Dr. Burch is ideal, safe and simple. I feel that we are greatly indebted to him for his trouble in putting it before us.

In children with approximately 15° of convergent squint the Jameson recession operation is good, there is little disturbance of the muscle covering, the reaction is slight, and since gut is used there are no sutures to remove—a most important consideration. Combined with the tucking operation, when this is necessary, it makes an ideal operation. When a tucking is done and an operation on the opposing muscle is required, the recession operation is to be preferred to tenotomy. The latter may be uncertain in its effects, whereas the former is not.

Dr. Frank E. Burch, closing: We have particularly avoided consideration of any methods of muscle shortening other than our own technique for tucking. We owe Dr. O'Connor an apology for making the statement in this paper that the tucking operation is the *only* method which preserves the original insertion; that was entirely an oversight on our part. There are several typographical errors in the text in the Presession volume. One is the interchange of the words "internal" and "external" in the series of examples which are given in the procedures suggested for various types of muscle imbalance and deviation. These are, of course, only approximate, are not meant to be arbitrary or exact, and I hope they will be accepted in that spirit. They were suggested from thorough effort to analyze our experience.

The principles we have mentioned in correcting deviations are in accordance with the principle in muscle dynamics so clearly described by Ronne in 1927 in the Archives of Ophthalmology. The amount of deviation which may be corrected by tucking a single muscle is rarely over 20° or 25° at most. Dr. Shannon recently said he was able to correct 35° by the tucking of one muscle alone. I doubt if we should attempt that because of possible trauma.

Dr. Davis asks about the wearing of glasses. We consider this relatively unimportant. If a patient with a squint has been wearing glasses which correct it he should continue to wear them. As to best age for operation, I can only say that the tucking method offers opportunity for safer operations at an earlier age than any other we have used.

The question has been raised, what becomes of the buried silk? We do not know. For three years we used catgut, and we know it produced large granulomatous humps which were unsightly and slow of absorption and annoyed us a good deal. We then tied the silk outside the conjunctiva for four years, until Dr. Greenwood taught us it was perfectly safe to bury the silk, and we have done that for one year. We do not know what becomes of it, but at the University of Minnesota we have started experiments to find out whether there is any difference between rayon silk and silkworm silk in its solubility.

I wish to thank Dr. Lancaster and the other discussors of this paper.

TAY-SACHS DISEASE.

Part I. Tay-Sachs Disease and Its Allied Conditions, with a Brief Review of the Literature.

Joseph L. Gouterman, M. D.,

Philadelphia.

There is a prevailing impression among those of the profession that Tay-Sachs disease is an affection of infancy, under the age of six months; racial in character, occurring only in the Jewish race, and rare in its incidence. A review of the literature on the subject militates against this impression. It will be the aim of this paper to show that amaurotic family idiocy is quite prevalent, having passed the stage of keeping account of the number of cases reported; covering a span of life between the ages of three months and thirty-five years; and not especially either race or sex selective. If this compilation should prove of some assistance to the ophthalmologist and neurologist in a more correct orientation among the obscure cases of juvenile and adult optic atrophies and amblyopias, I would consider this effort amply justified.

Historical Data.[1]

The first published account of the disease was rendered by Tay,[6] in 1881, under the title "Symmetrical Changes in the Region of the Yellow Spot in Each Eye of an Infant." In 1887 Sachs published a paper on a case of blindness associated with idiocy, entitled "Arrested Cerebral Development." Nine years later Sachs recognized the familial element in the disease and coined the name "amaurotic family idiocy," quoting Higier as being the first to propose the name of Tay-Sachs disease. Ward Holden,[2] who made a pathologic report on the eyes of Dr. Hirsch's patient, is credited with the discovery of the relationship between the fundus picture and the general nervous affection. The first cases of the juvenile amaurotic family idiocy were described by F. E. Batten.[3] It was Vogt[1] who, in 1905, gave the name of juvenile type to those cases in which the disease began in youth instead of in infancy, without showing a predilection for Hebrews.

Age Incidence.[1]—

Just after the first year—Dr. Findlay's three cases.
During the second year—Third child in Vogt's second family.
During the third year—F. E. Batten's five cases (1914).
During the fourth year—Bielchowsky's four cases.
During the fifth year—Ichikawa's case.
During the sixth year—Firucowa's two cases.
During the seventh year—Mayou's three cases (1904).
In 1903, Batten reported 2 cases at the age of 6.[2]
In 1906, Spielmeyer reported four cases at the age of 6.
In 1907, Oatman reported two cases at the ages of 6 and 7.
In 1907, Vogt reported several cases.
In 1909, Wandless reported three cases.
In 1910, Rogalski reported one case at the age of 7.
In 1910, Kuffler reported four cases at the age of about 6.
In 1912, Gifford reported two cases at the age of 7.
In 1912, Schob reported one case at the age of 6.

In the literature we find cases reported between the ages of 7 and 35.

SYMPTOMATOLOGY.[1]

Infantile Tay-Sachs Disease.—

1. Mental impairment, during the first months of life, leading to absolute idiocy.
2. Paresis, or paralysis of the greater part of the body, which may be either flaccid or spastic.
3. Normal, deficient or increased reflexes.
4. Diminution in vision, terminating in absolute blindness (the cherry-red spot in the region of the macula lutea, and later, a simple optic atrophy).
5. Marasmus and a fatal termination, as a rule before the age of 2.
6. Occurrence of the affection in several members of the family.
7. Nystagmus in some but not in all of the cases.
8. Strabismus in some but not in all of the cases.
9. Hyperacusis in some but not in all of the cases.
10. Convulsions in some but not in all of the cases.

Add to this the fact that the children are apparently in perfect health at birth and generally remain so until three to six months, also the racial predisposition (Jews) and one has a fairly complete picture of the disease. Recently Pelizaeus[4] described a condition, called aplasia axialis extracorticalis congenita, or Pelizaeus-

Merzbacher's disease, coming on in older children, which somewhat closely resembles amaurotic family idiocy, although rarer in its incidence, and generally appearing at about three years of age. The children live longer than in the Tay-Sachs variety, and may attain adult life. There are optic atrophy and blindness but no red spot develops. There is also a progressive spastic paralysis which is diplegic in distribution. These cases show a widespread change, chiefly of the axis cylinders, and are thought to be due to a congenital defect of formation, or agenesis of the axis cylinders, especially of the long extracortical association fibers, while the familial or Sachs type is rather a primary cellular degenerative process. Nystagmus and tremor are frequently symptoms in the Pelizaeus form.

Juvenile Tay-Sachs Disease.[3]—The children are born healthy and develop normally to a period of life varying with the different families. It always attacks the members of any one family at the same time of life, either at the age of six to eight years or at the age of fourteen to twenty years. They begin to show, first, loss of intellectual faculties; then loss of vision, and lastly loss of motor power. The disease progresses slowly, eventually rendering the child blind, demented and spastic. The disease is neither race nor sex selective. The change at the macula consists of a diffuse pigmentation, which extends more or less into the surrounding retina. The disc is of a grayish-white color, with vessels a little smaller than normal.

Oatman[3] subdivides this variety of Tay-Sachs' disease into—

1. The "maculocerebral" type, in which both retina and brain are attacked by the pathologic process, resulting in failure of vision combined with failure of intellect. The onset of the disease is between the fifth and seventh years—the period of second dentition.

2. The "macula" type, in which the retina is affected by the process, but the brain escapes. Hence visual disturbances without dementia. The onset of this variety is between 14 to 16 years—the period of puberty. Important[1] biochemical changes take place at the periods of infancy, second dentition and puberty, such as accumulation of the Nissl substance in the nerve cells, deposition of myelin around the axial fibers of the neurons, particularly in the brain cortex, where there is scarcely any present at birth. Thus if a breakdown in the nervous system is imminent, the proba-

bility is that it will occur during these periods rather than in the intervening periods.

Ocular Manifestations of Tay-Sachs Disease and Its Allied Affections.—External findings: Pupils are dilated and sluggish in their response to light, perhaps in some instances completely irresponsive. Nystagmus, strabismus and oculomotor paralysis have been observed in some cases.

Ophthalmoscopic Features of the Infantile Type of Tay-Sachs Diseases.—Macular phenomena—the earliest to appear—consist of a cherry red spot at the fovea, surrounded by a broad grayish-white area, about twice the size of the optic disc, not extending to the papilla, a picture that resembles an occlusion of the central artery. This condition, however, is excluded by the darker cherry red spot, not present at birth, and remaining unchanged from its advent until death, while that of occlusion disappears within from ten to fourteen days, and by the absence of any opacity about the papilla.

In the atypical forms of amaurotic family idiocy, especially in the late infantile types, instead of a cherry red spot other appearances may be found in the fundi which might be considered as equivalents, namely, grayish discoloration and a nebulous appearance of the retina (Hassin and Pamerlee's case); a slight veil-like, milky-bluish haze of the macula gradually fading into the surrounding color of the retina (Koller's case); a reddish macula surrounded by a dark pigment ring (Muhlenberger's case).

Changes in the Papilla: The optic nerve, though not at first affected, later on becomes pale or even quite white, but not yellowish as optic atrophy sets in with ultimate complete blindness. The atrophy is of the simple variety.

The retinal arteries are contracted and smaller than normal.

The retina finally undergoes depigmentation.

Field characteristics: A central scotoma may appear early for green and red with normal peripheral fields for white. There may be early failure of central with preservation of peripheral vision. This may be preceded by a history of good vision.

In the juvenile and adolescent types of amaurotic family idiocy the pigmentary alterations in the region of the macula may be but slight. In the typical type the macula shows an irregular, round, reddish-black spot, surrounded by peppered pigmentary changes.

The optic disc is pale. The retinal vessels are small (F. E. Batten).

ETIOLOGY.

All writers[6] on the subject are in accord with the statement that there is no evidence of inflammatory changes in the affected parts and that the disease is due to a primary change in the cells themselves. Gordon Holmes showed that the changes were not due to an arrest of their development or to any bacterial toxin. Sir Frederic Mott attributed the cellular changes to a failure in the inherent specific trophic property of the nucleus of a nerve cell, upon which the vitality of the whole neuron depends. Waren Tay[7] regarded the disease as a congenital condition, the pathologic changes being confined to the retina. Sachs[8] first described this entity as a congenital, genetic, morbid process—*agenesia corticalis*. In later publications he combined the above with a degenerative or abiotrophic change. Collins and Mayou[9] view this disease as an abiotrophy. Sir Herbert Parsons[10] disregarded the abiotrophic theory, believing this condition to be the result of some inherent biochemical property. Of all the suggested theories, the one most generally favored has been the presence of autotoxins (Stock, Stargardt, Schlapp), which, under the stress of second dentition, attack the retina and thus initiate a progressive degeneration of the neuron system.

Predisposing factors:[11] (a) Familial tendency, inasmuch as frequently several children in the same family are affected.

(b) Race. It is more common in the Hebrew race, but by no means confined to this race.

(c) Blood relationship of the parents.

(d) Trauma to pregnant mothers.

(e) Neurotic taint.

(f) Premature arteriosclerosis (Pusey).

(g) Syphilis.

(h) Toxins resulting from exanthemata.

(i) Toxemia of child. J. Epstein suggested as a possible cause for Tay-Sachs disease gastrointestinal toxemia, resulting from abnormal metabolism of calcium salts, initiated by a dysfunction of the parathyroids. G. Marinesco explained that pathologic changes in the intracellular ferments might be responsible for the disease. In a later publication he stated that the course of the

capillaries is strewn with glycogen granules and that an intimate relationship seems to exist between the gravity of the lesion and the deposit of glycogen.

HISTOPATHOLOGY OF TAY-SACHS DISEASE.

Schaffer[12] divides the pathologic changes into two phases:

(a) The *swelling phase*, characterized by swelling of the hyaloplasm of the neurons. The nerve cells may swell as a whole or in part, as in the dendrites of axons, which may be swollen like a balloon in a localized area. The extent of the swelling is a distinctive feature of the histopathologic picture of the disease which is not found in the exogenous diseases. The edema affects the ectodermal elements, while the mesodermal elements—membranes and blood vessels—remain unaffected.

(b) *Precipitating phase*, in which granules appear in the swollen hyaloplasm, followed by round or polygonal bodies, which stain with hematoxylin and which fill the gaps. These gradually developing bodies are prelipoid degenerative products, arising from the hyaloplasm. The lecithinoid bodies are partly carried away by glial cells via the perivascular lymph spaces, and partly transformed into fats within the cells. The nerve cells are relatively more affected than the neurofibrils, the latter being attacked secondarily. According to Collins and Mayou,[9] the essential histologic features of amaurotic family idiocy are:

1. Progressive loss of Nissl substance in all the neurons in the body, and (2) the increase in the neuroglial fibrillar substance to an abnormal degree.

Chemical examination of the brain by Sidney A. Mann has shown:

(a) A decrease of nucleoproteids, which may be associated with the disappearance of the Nissl substance in the neurons.

(b) And the increase of the simple proteids, which may be correlated with the increase of glial fibers.

In view of the pathologic findings in the ganglion cell layer, the macroscopic fundus picture can be explained on anatomic grounds. In the normal eye the ganglion cells vary from 10 to 30 microns in diameter. In the periphery, where the cells are scattered, large cells abound; but in the macular region, where the ganglion layer is several cells deep, the cells are smaller, irregularly pear-

shaped and packed closely together. In the fovea the ganglion cells are absent. At the margins of the fovea the ganglion layer is six to ten cells deep. One disc diameter from the fovea horizontally it is three to four cells deep; two disc diameters, two to three cells deep, till at the periphery it is a scattered layer of single cells. In the vertical meridian the ganglion layer thins out most rapidly. The alteration in size and shape of the ganglion cells in amaurotic family idiocy gives the snowy white appearance to the retina in those areas where the ganglion cells are numerous —that is, one to three disc diameters around the fovea. The fovea proper, having no ganglion cells, is not altered in color and hence stands out as a vivid red in contrast to the surrounding white retina.

TAY-SACHS DISEASE AND ALLIED CONDITIONS.

Amaurotic family idiocy, retinitis pigmentosa and macular pigmentary degeneration, whilst possessing group characteristics, yet appear to have fundamental similarities, as if the same etiologic and pathologic processes permeated them all.

Retinitis Pigmentosa	*Macular Pigmentary Degeneration*	*Amaurotic Family Idiocy*
AGE AND PROGRESS		
Commences early in life and progresses slowly.	May commence at age of 6 or not until the 2nd decade, rapid failure of sight and then stationary.	Onset at age of 3 to 6 months; rapid failure of sight; death in two years.
HEREDITY		
More than one generation affected; no racial proclivity.	Familial, no racial proclivity.	Familial; only met with in the Jewish race; no racial proclivity in the juvenile or adolescent group.
VISUAL DEFECTS		
Nyctalopia, followed by annular scotoma; central vision last affected.	Central scotoma; no contraction of fields; no nyctalopia.	Central loss of sight early, progressing to complete blindness.
CONDITION OF RETINA		
Primary affection of bacillary layer at equator, later formation of branching patches of pigmentation.	Primary affection of bacillary layer at macula, later formation of dots of pigmentation.	Primary affection of ganglion cells, causing white opacity around macula.

GENERAL NERVOUS SYSTEM

Sometimes mental weakness; cause unknown.	Sometimes dementia; ganglion cells affected as in amaurotic family idiocy.	Always dementia; all ganglion cells in body involved.

Differential Diagnosis:[13] We should differentiate these conditions from congenital defects, inflammations of the retina or of the choroid, and other forms of retinal degeneration. Congenital macular defects differ from Tay-Sachs' disease in that they do not appear after a child has enjoyed good vision, do not progress or induce optic atrophy and are not associated with a progressive loss of intellect or of muscle power. A central retinitis is excluded by the pigmentation of the retina, while in central choroiditis the black and white spots in the macula are not enclosed in a defined oblong, and are not likely to be associated with a dusky pigmentation of the retina, such as is common in this disease or these diseases. When the choroidal markings are visible within the affected area we have to deal probably with a choroiditis or a sclerosis of the choroid.

The history of previously good vision and the absence of symptoms of hereditary syphilis suffice to show that the dusky pigmentation of the retina is not the same as that of the snuff colored fundus.

Pigmented degeneration of the retina is excluded by the absence of hemeralopia, the early failure of central with preservation of peripheral vision, instead of the reverse, the early appearance of a central scotoma and the white rather than the yellowish color of the papilla.

The picture presented in circinate degeneration is different and this condition seldom is met with in childhood.

Treatment: General hygienic measures, calcium bromide and parathyroid extract should be tried in the early cases. The progress of the disease is rarely arrested by the symptomatic treatment.

SUMMARY.

The various types of Tay-Sachs disease have features in common, namely: The patients are born healthy, fair-skinned and comely, from healthy parents, usually related to each other.

This affection is familial, hereditary, bilateral, having the same histopathology, prognosis and lack of specific treatment.

Distinctive features: Infantile type occurs at the age of three to six months; it is race selective (Hebrew), and is characterized by visual disturbances, macular changes (cherry red spot), leading to blindness, loss of muscle power, dementia and death at the age of two years.

Juvenile type develops at the age of five to seven years; it is not race selective and possesses the same visual, mental and motor disturbances with the exception of a slower progress of the development of the symptomatology, brownish pigmentary macular changes instead of a cherry red spot, and death taking place at the age of about twelve years.

Adolescent variety occurs at the age of fourteen to sixteen or twenty years; it is not race selective; shows visual disturbances with macular changes similar to those found in the juvenile type and a central scotoma; loss of muscle power, but no dementia. This variety of Tay-Sachs disease is not fatal.

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Part II. Histopathology of Two Cases of Tay-Sachs Disease.

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Philadelphia.

Neurologists have devoted considerable time to the familial diseases, because of their bearing on the entire problem of nervous conditions. In fact, Karl Schaffer of Budapest has devoted the last twenty years to these problems, the results of which are

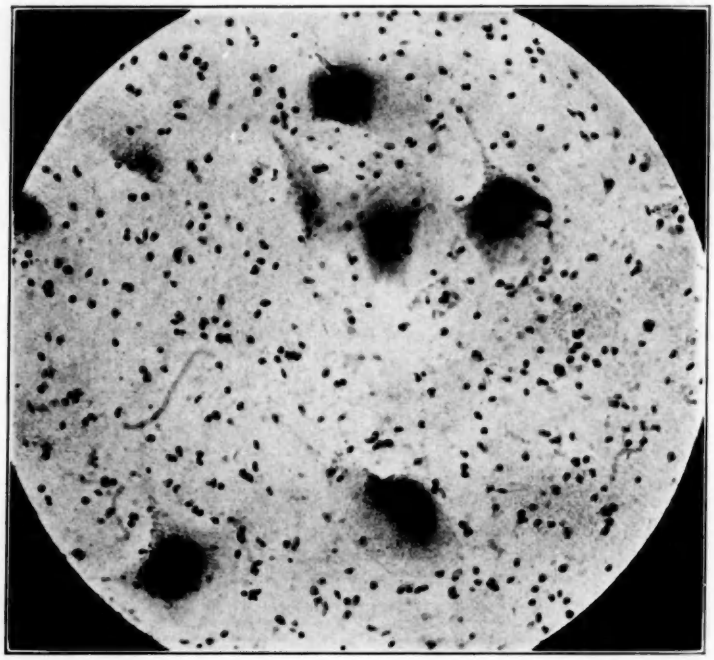

Fig. 1. Typical changes in the ganglion cells of the anterior horns of the spinal cord. (Schaffer's cell disease.)

embodied in a monograph.[1] The individual articles dealing with this subject are so numerous that it is impossible to quote even a small portion of them.

One of the best known and most sharply defined of this great group of hereditary diseases is the infantile form of amaurotic idiocy or the so-called Tay-Sachs disease, or better Tay-Sachs-Schaffer's disease, because while Tay first described the charac-

teristic ocular findings, and Sachs the clinical picture, it was left for Schaffer to first describe the characteristic and pathognomonic ganglion cell changes in the entire central nervous system.

The clinical picture is usually so characteristic that little doubt is present in the mind of the examiner that he is dealing with this condition. Given a child of Jewish parentage who has developed normally until the age of six or eight months and then begins to show an increasing weakness of the extremities up to complete paralysis with increased muscle tone and hyper-reflexia, with gradual mental deterioration, an eye ground examination is

Fig. 2. Bielschowsky stain to show neurofibrillar swellings and irregularities, and the changes within the swollen ganglion cells.

urgently demanded. The characteristic cherry red spot in the eye ground is usually the final word in the diagnosis of this condition. Blindness is the eventual result.

Death takes place before the second year. It might be of interest to give a short sketch of the pathology at this point and to elaborate on it in the description of our cases. Briefly, the gross findings are usually developmental defects, with a peculiar hardness of the brain, that is commented on by all recent authors, and the presence of very small and very large convolutions. Microscopically there occurs a swelling of the ganglion cells and

their processes of the entire nervous system of a type that is characteristic only of this condition. The retina, as part of the brain, shows the same type of reaction.

We have two typical cases to report. The first concerns a Jewish girl who was fifteen months of age at the time of death. She was sent to the hospital because of convulsions. There was a history of mental disease on both sides of the family. Another child had had a similar condition at the age of seventeen months, and the condition was diagnosed hydrocephalus; postmortem

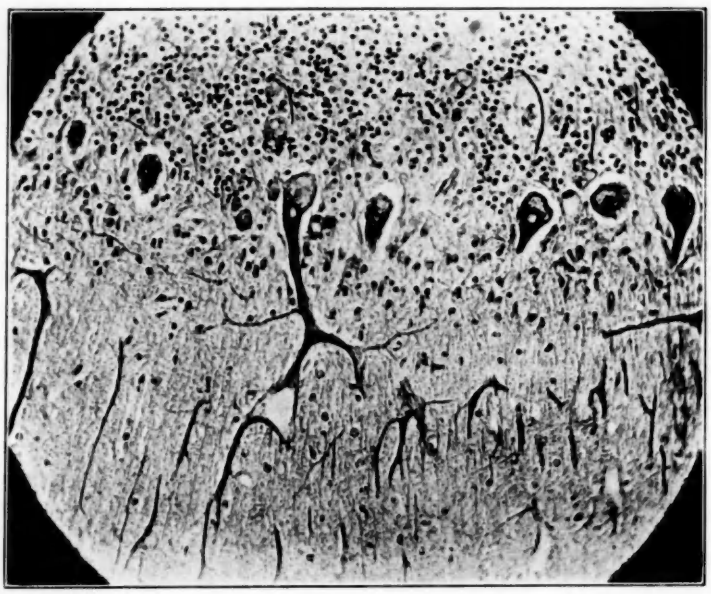

Fig. 3. Bielschowsky stain of cerebellum. Ballooning out of Purkinje cells, as well as of processes can be made out very readily.

studies were not permitted. The patient was a full term child and was breast fed for two months. At the third or fourth month it was noted that she was unusually sensitive to sounds, starting at the least noise. At six months the mother noted that the child's head was larger than normal, and at this time the child could not handle herself as well as a normal child. Convulsions began at about three months. On examination, the child appeared well developed, but her head measured 18½ inches (46.99 cm.) in circumference. There were convulsive movements with deviation

of the head and eyes. The spinal fluid was under increased pressure (25 mm.). The eye grounds showed the characteristic cherry red spot. The extremities appeared somewhat flaccid, but the reflexes were slightly increased. Little if any vision was present. Progressive emaciation occurred, with irregular fever and death ten weeks after admission.

At autopsy the brain was found to be larger than normal and was as firm as if it had been in formaldehyde for weeks. The convolutional pattern was peculiar in that great variation in the size of the individual convolutions occurred, some being very small and irregular and others very large.

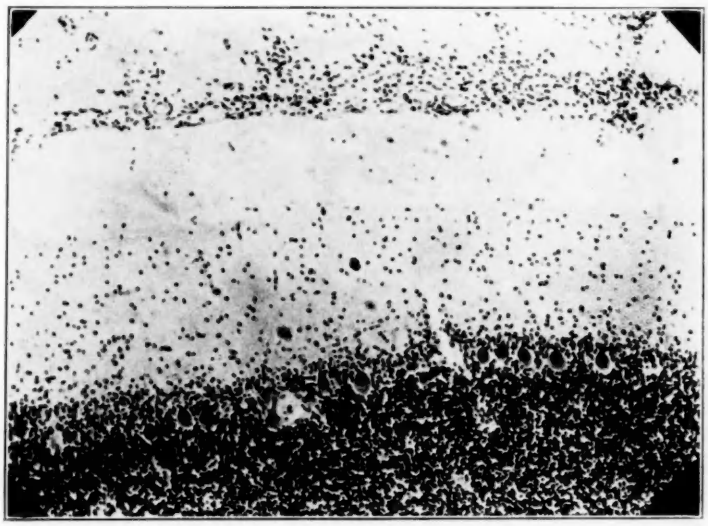

Fig. 4. Cell stain of cerebellum. Note intense swelling of Purkinje cells that are fairly numerous. Granule layer normal.

Microscopic examination showed as follows: The pia-arachnoid was definitely thickened as result of edema plus fibroblastic proliferation, in the meshes of which were occasional macrophages but no inflammatory elements. In the cortex the outstanding feature was the involvement of every ganglion cell, large and small, by the Schaffer cell disease. You, as ophthalmologists, are not greatly concerned with the finer cell changes, so suffice it to say that Fig. 1 gives the appearance of this type of cell change in the usual toluidin blue alcohol fixed preparations. There is a

peculiar swelling of the cell body, as well as its plasmatic processes. So great may the swelling be that the cell itself and even its processes may resemble small balloons. There is a disappearance of the normal Nissl body accumulation in the cell body, so that in the final stage one sees a perinuclear collection of dust-like particles as the remains of the previous stainable substance, while the rest of the cell body shows a meshwork, in the individual spaces of which can be demonstrated by appropriate staining methods a lipoid substance. As a rule, the nucleus is gradually displaced until it assumes a peripheral position. It is remarkable that the nucleus itself shows but mild changes in comparison to the cytoplasmic alterations.

Fig. 5. Spinal cord—Myelin sheath stain. Shows lack of myelin in pyramidal tracts. (Patient had never walked.)

With the neurofibrillar stain of Bielschowsky (Fig. 2) one can readily note the balloon shaped swelling of the ganglion cells, as well as changes in the plasmatic processes.

One notes the meshwork within the cytoplasm with the intracellular neurofibrillæ pressed around the margins of the cells, most of which eventually disappear. The changes in the nerve fibers are well shown in this picture, while the axonal changes in relation to the ganglion cells are well illustrated in Fig. 3 of the cerebellar pathology, where a balloon-like dilatation of a process can be seen in relation to one of the Purkinje cells.

It will serve our purpose to state briefly that the intercellular tissue did not remain passive. Phagocytic cells were present in abundance, while specific stains showed also a proliferation of fiber forming astrocytes, so that it can be considered as an actual sclerosing process. It is probably this factor that gives to the brain its hardness.

The blood vessels themselves have undergone but little change. It has already been mentioned that the changes in the ganglion cells and the glial proliferation belong to a universal process. Hassin has felt that the thalamus is the seat of the most intense changes. This is entirely possible in view of the characteristic clinical features.

The second case is one that has only recently come to our laboratory. The clinical history was so characteristic that a diagnosis could be made on that alone. The findings entirely confirmed the diagnosis. Microscopically the picture was so typical that anyone at all acquainted with the findings could make the diagnosis at one glance.

It might be of interest to mention another case, which is to be reported in detail in a future contribution. It has recently come to be recognized that abortive or incomplete cases of so-called amaurotic idiocy can occur. This is well illustrated by a case of a man of nineteen who died in status epilepticus and in whom no suspicion of the underlying cause was to be found clinically. He appeared to be the ordinary so-called "idiopathic" type of epilepsy with marked mental deterioration. The eye grounds showed but a mild optic atrophy. The microscopic study of the brain of this patient gave us our first clew as to the underlying pathology. It showed us the typical Schaffer-cell disease practically entirely limited to Ammon's horn. Cases of this sort, while rare, are not unknown.

In conclusion, it is to be emphasized that while ganglion cell changes, for the most part, are not specific of a disease process, we do have a notable exception in the Schaffer cell change, which is pathognomonic of a group of diseases that go under the general term of amaurotic idiocy, of which the infantile form was the first described and the best known.

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DISCUSSION.

Dr. Luther C. Peter, Philadelphia: Three phases of this timely presentation are of practical importance to us as ophthalmologists: First, the classification; second, the pathology; and third, the etiology.

Although the clinical diagnosis can be made without the eye findings, the neurologist as a rule asks for an eye report to complete the clinical picture. In the infantile variety of the disease the eye symptoms are definite, and in themselves are diagnostic of amaurotic family idiocy.

From a more or less confused accumulation of observations as to the symptomatology of Tay-Sachs' disease, order has been brought out of chaos by a classification of the disease into three groups, namely, the infantile, the juvenile and the adolescent types. The first occurs in apparently normal infants of Jewish parentage, is marked by paralysis, idiocy, optic atrophy, a cherry red spot in the macula surrounded by a pale zone, and terminates fatally, as a rule, before the end of the second year. The diagnosis in this group offers little difficulty.

The second, or juvenile, type is not race selective. It begins in apparently healthy children, from six to eight years of age, with mental deterioration, visual loss associated with atrophy and macular degeneration, and finally motor disturbance. The disease terminates fatally, as a rule, before the twelfth year. The cherry red spot is not in evidence but is replaced by varying degrees of macular pigmentation.

The third, or adolescent, type has its onset between the fourteenth and twentieth years. It is not race selective. Visual disturbance and motor weakness are the same as in the other types, but mental deterioration is not found. It may not be fatal. This group is in need of further study to place its diagnosis in the clear-cut category of the infantile and juvenile varieties. The one definite phase of the adolescent group it shares with the other types, namely, the pathologic findings.

Winkelman, Schaffer and others feel that the pathologic findings are the same in each group, and so characteristic that Winkelman believes the Schaffer cell disease is a condition which is only found in amaurotic family idiocy. If these facts are correct, further studies will finally classify the clinical aspects of the adolescent type. The last case to which Winkelman refers, a case which has not as yet been fully reported, raises the question, which he intimates, as to whether the adolescent type really is a third group of Tay-Sachs' disease or an abortive form of the juvenile type. This fact awaits further confirmation.

The final phase of practical importance is the etiology of the disease, a phase which is as yet in some doubt. Many theories have been advanced, but most of them are lacking in proof. The autotoxin theory, which the essayist feels has received most support, falls short in essential respects. It is possible that autotoxins may play a rôle as precipitating factors, but why should these toxins be race and family selective? Each type occurs in apparently normal individuals. The abiotrophy theory of Collins seems to the speaker to fit in with known facts better than any other theory advanced. From our present knowledge, we have no means of preventing the disease, and nothing is known by which the disease can

be arrested. Furthermore, the onset of the disease seems to be at such periods of the life of the individual when special demand is made on the personal economy that an absence or difficulty in vital cell energy marks the beginning of the abiotic atrophy.

The disease is not one which is frequently observed, but every ophthalmologist who is connected with hospitals of fair capacity is called upon with sufficient frequency to cooperate in the study of the condition to make it essential that he should be familiar with the important facts which were presented by Dr. Gouterman in his interesting contribution.

Dr. Parker Heath, Detroit: Pick and Bielschowsky have recently noted an association between Niemann-Pick's disease, which is a familial lipoid disease of the infant, and Tay-Sachs' amaurotic family idiocy. Rowland has recently correlated the pathological and clinical findings found in Niemann-Pick's, Gaucher's and the Schuller-Christian syndrome, all manifestation of lipoid-histiocytosis. The speaker, at the recent American Medical Association Section meeting, suggested that a number of little-understood fundus lesions which exhibited familial and constitutional tendencies be grouped under a single head of basic lipoid pathology.

There have been many studies of the pathology of Tay-Sachs' disease, including those of Hassin-Schaeffer, largely confined to the nerve tissue. These studies are very much in agreement in the matter, but Schaeffer feels that the Tay-Sachs' syndrome is a purely ectodermal, and not concerned with the mesodermal reticulum. However, lipoids are found extensively in many of the cell changes, both early and late. It seems fairly well accepted that the pathological changes present in this syndrome, as described in juvenile, infant and adult forms, are practically identical. It is a familial disease characterized by peculiar lipoid storages or degenerations of the ganglion cells, apparently constitutional, and in the infantile form, racial.

In searching for etiology we are handicapped because studies of the living are incomplete. Studies after death have been made largely on nerve tissues. Further work should be directed along lines showing chemistry of the blood stream as well as complete necropsy studies. It is likely that lipoid excess will be found present in other tissues than ganglion cells in this tissue. The speaker therefore would urge those treating patients with Tay-Sachs' disease to make repeated blood chemistry studies of the living, and in the event of death of the patient to study the postmortem material thoroughly.

Dr. Isadore Goldstein, New York City: My remarks are with reference to Niemann-Pick's disease which Dr. Wexler and myself have recently studied.

Niemann-Pick's disease is a familial disease of a certain type. The symptoms are those of a distended abdomen due to an enlarged liver and spleen. The skin is light brown. The blood smear presents a large number of vacuolated lymphocytes. The cherry red spot at macula is similar in nature to that seen in Tay-Sachs' disease (amaurotic family idiocy). The changes in the disc, however, differ in Niemann-Pick's disease. They are yellowish, due to the deposits of lipoid in the nerve, while in amaurotic

family idiocy they are usually atrophic. The pathology is that of a general histocytosis. There is disturbance of lipoid metabolism. Most cells have lipoid in them; the excess of lipoid is taken up by the histocytes which appear as the so-called foam cells. Few of these foam cells are seen in the retina. The changes in the ganglion cells are the same as in amaurotic family idiocy, except they are more severe. Through the choroid foam cells are encountered frequently, some of which contain pigment. Throughout the sclera many foam cells are seen. The ganglion cells accompanying the ciliary nerves show the same changes as those occurring in the brain.

DR. J. I. GOUTERMAN, closing: I wish to thank Drs. Heath and Goldstein for bringing up the question of probable relationship between Gaucher's, Niemann-Pick's and Tay-Sachs' disease. It was not included in my paper because this new angle is not universally accepted as the possible etiology of Tay-Sachs' disease.

According to Bielchowsky, the pathogenesis of amaurotic family idiocy must be linked with the source of lipoid substance formed within the cells. This phase was more elaborately worked out by Bloom, Niemann and others.

In 1914, Pick reported seven cases of the Niemann-Pick type, in which he made a complete examination of all the organs, including the central nervous system, he also recorded that one of these cases was easily recognized as a form of the Niemann-Pick type, in which all of the cardinal symptoms of the Tay-Sachs' variety were present. It is Pick's contention that in Gaucher, Niemann-Pick and Tay-Sachs diseases the same disturbance in lipoid metabolism is the predominant factor. When this metabolic disturbance affects the entire body, Gaucher's or Niemann-Pick's types are developed. The former might be called the juvenile and the latter the infantile form of splenohepatomegaly or lipoid histocytosis. The Tay-Sachs' form is a disturbance of the same kind restricted to the nervous system.

THE ESTIMATION OF THE TOTAL REFRACTIVE ERROR WITHOUT A CYCLOPLEGIC.

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GENERAL INTRODUCTION.—The fact that we discuss "Refraction Without a Cycloplegic" seriously does not imply that any of us can ever hope to do without cycloplegia altogether. It implies only that we are becoming aware that noncycloplegic refraction can and should be done much better than we have been doing it, both in postcycloplegic refraction and in those cases in which, for any reason, a cycloplegic does not seem necessary or wise. This paper should, therefore, be considered as a contribution to the "Science and Art of Noncycloplegic Refraction," and in no way as an argument against the use of cycloplegia. Noncycloplegic refraction should replace cycloplegia in the practice of any man only so far as he finds his noncycloplegic refraction accurate enough to do so safely. This will not be very far unless he really understands accommodative control by overcorrection as well as by cycloplegia, and unless he uses each to check the weaknesses of the other.

It is almost axiomatic that accuracy in the measurement of refractive errors is not possible without the elimination of practically all accommodative interference. This can only be accomplished by efficient control of the accommodation, either by cycloplegia or by overcorrection, which are the only ways of controlling accommodation. The control by cycloplegia is always efficient if cycloplegia is proven to be complete by the recession of the near point to infinity under full correction. Unless so proven, it is much more often inefficient than most of us appreciate. The control by overcorrection, as usually employed, is frequently inefficient, and it is the purpose of this paper to discuss the question of how control by overcorrection may be employed so as to be as dependably efficient as possible. This requires much more than what we know as simple or bilateral fogging. It requires a sound knowledge of the fundamental principles of control by overcorrection, knowledge of the many small but

vital details of the application of those principles in practice, and the thoughtful use of this knowledge by the eye physician who has had good cycloplegic training. It also requires the use of absolutely different methods from those used in cycloplegic and in manifest refraction, since most of those well known methods stimulate accommodation to some extent, and so defeat full control by overcorrection. When those methods are used in bilateral fogging without such radical changes as the principles of efficient control by overcorrection demand, they result only in a partial accommodative control, which sometimes uncovers little more than the manifest error. The most important changes in these methods necessary for full control by overcorrection are: Skiascopy must always be done with a distant object of fixation and with both eyes overcorrected. In the subjective tests both eyes must be tested together at all times, the one not under immediate test being excluded by enough more overcorrection to give it slightly poorer vision. Overcorrection must be continuous and without the slightest break. Sharp vision must be constantly prevented in either eye except at the moment of finally determining the full error for that eye. With the maximum spherical error cannot be brought out by sharp vision, it can be brought out and measured only by skiascopy and by blurred vision. These methods are almost as radically different from those ordinarily used in bilateral fogging as they are from those used in cycloplegic and manifest refraction. Unless, however, we restrict ourselves to such methods as do not in any way limit full accommodative relaxation under overcorrection, we cannot hope to obtain real noncycloplegic accuracy with any frequency or dependability. Obvious as this is when we stop to think about it, most of us have not thought much about it. We are so accustomed to the use of our ordinary methods, which can ignore accommodation safely only when it has been eliminated by cycloplegia or by age, that we use them without thinking for all noncycloplegic refraction. Of course such noncycloplegic refraction is unreliable, and accuracy occasional or accidental, except in the easiest cases. But it is the way in which the great bulk of noncycloplegic refraction is still being done.

While the principles and methods of efficient accommodative control by overcorrection are not yet well described in the literature or taught in the textbooks and schools of ophthalmology, most of them are known and used by such ophthalmologists as

have worked them out for themselves. These ophthalmologists have found accurate noncycloplegic refraction possible in a very large number of cases. Accuracy is limited, however, to the men who seriously try to obtain it, who check it by frequent use of a cycloplegic, and who thus learn how to recognize the cases in which it is not obtainable.

General appreciation of these facts is further hampered by the connotations of an outworn terminology. Every method of refraction, new and old, except that of cycloplegia, has to be classed as "refraction without a cycloplegic." That term now includes manifest refraction, simple, bilateral and balanced fogging, combined skiascopy and fogging, and cyclodamia, each more accurate, and accurate in more cases than its predecessor in the list. These are all specific methods which must be definitely distinguished, and each one analyzed and studied from the point of view of the amount and certainty of its accommodative control before we can understand or appreciate it, or compare its potential or practical accuracy with that of cycloplegia. The term "refraction without a cycloplegic," having to include all these methods, has lost almost all specific meaning. The old connotation of essential inaccuracy attached to it was justified by its methods of manifest and simple or bilateral fogging, but is not justified by its newer methods which so often control the accommodation efficiently. The old connotation, however, still leads many men to think that the newer methods must be essentially inaccurate simply because they are "noncycloplegic," sometimes even without making any effort to find out what the newer methods are. It is small wonder, therefore, that "refraction without a cycloplegic" has come to be so confused, misunderstood and controversial a subject, and that its general discussion has so often been injudicious and futile.

Under such conditions, it is with considerable trepidation that, in response to the urgent request of your secretary, the writer has here attempted to clear up some of the misunderstanding about the newer noncycloplegic refraction, and has ventured to discuss the fundamental principles of efficient accommodative control by overcorrection and to suggest a method of applying those principles which has proved much more accurate in his experience than other noncycloplegic methods. He has endeavored to present the necessary facts and his conclusions therefrom as briefly as is compatible with clearness, yet the field is so large

and ill explored that the paper is a long one. It, therefore, seems best to present only this general introduction in the meeting, and to leave the paper as printed in the presessional volume to speak for itself.

Two sincere objections to any discussion of noncycloplegic accuracy at this time must first be briefly considered.

The first objection is that noncycloplegic accuracy is unnecessary, since we can always obtain accuracy more simply and surely with cycloplegia. This is the objection of that part of the ophthalmologic profession which uses cycloplegia routinely, but does such noncycloplegic refraction as has to be done with little or no control of the accommodation or no more than is obtained by bilateral fogging using cycloplegic methods. Their use of cycloplegia is often beyond criticism when they prove its completeness, but the faults of their noncycloplegic work are not always appreciated. 1. Such noncycloplegic refraction is quite often unnecessarily inaccurate, and since everyone has to refract some patients without a cycloplegic the problem of doing it as well as it can be done should be faced squarely. 2. Postcycloplegic examination loses most of its potential value unless it makes use of the principles of efficient control by overcorrection. 3. Cycloplegia affords no adequate recognition of accommodative anomalies or of unusual accommodative habit, both of which should be allowed for in the correction ordered. 4. Neither cycloplegia nor manifest clearly reveal the momentarily excessive but poorly sustained accommodation so often seen in young presbyopic patients. 5. We almost always need more information about accommodative habit for satisfactory correction than is afforded by the difference between the static and the manifest errors. This information is most surely obtained under efficient control by overcorrection.

The other objection is that any discussion of noncycloplegic refraction should be avoided because it may give aid and encouragement to the optometrist, who may perhaps distort it for purposes of propaganda. This is the objection of that large part of our profession which puts more faith in limiting the partly trained than in demonstrating to the public the much greater value of full training. The general answer to this objection is that we have long since learned that it is usually far better to obtain, discuss and spread scientific knowledge than to try to restrict it; and that distorted science ultimately reacts against the

distorter. The specific answer is that much broader and sounder training is required to refract accurately without a cycloplegic than with one; that an essential part of that training consists in frequent cycloplegic check upon the way in which one uses the principles and methods of efficient accommodative control by overcorrection in his own work; and that experience has shown that medical training and real medical ability are usually required to make noncycloplegic refraction really accurate in any very large number of cases. Despite the fact that some optometrists are college graduates and have also been well grounded in physiologic optics, most of them lack knowledge of efficient accommodative control by overcorrection, and all of them lack the indispensable medical training which alone can fit them to relieve human ills safely and surely, even though those ills be mainly refractive. The problem is almost never that of the eye alone, but that of the eye in relation to the rest of the body, especially to the nervous system. The osteopaths have already learned that general medical training, even though obtained outside of the regular schools of medicine, is essential to their continued existence. The optometrists will also learn this. Without disrespect to the nonmedical attainments of the better trained and ethical optometrists, some of whom no longer try to delude the public into thinking of them as "doctors," it is still the opinion of the writer that the optometrist bears much the same essential relation to the fully trained eye physician as the midwife does to the trained obstetrician. Both lack general medical training, and so need and should have medical supervision, as well as strict legal control. Both have their necessary place in the community under present conditions, but only until the community has been taught by experience to demand better service than they are able to give, and until the eye physicians and obstetricians of the community are prepared to give that better service, and to give it at a price which the community can afford to pay. No optometrist, however, can effectually compete with the well trained eye physician who is a really good refractionist without, as well as with, cycloplegia. But he may be a serious competitor for the eye physician whose training in refraction has been inadequate; who does not appreciate that the use of a cycloplegic, necessary as it is in many cases, does not of itself make a good refractionist; and who does not use, in both his noncycloplegic and his postcycloplegic refraction, efficient accommodative con-

trol by overcorrection. If, as the writer sincerely believes, the ultimate solution to the problem of the optometrist lies largely in the better training of the eye physician in accurate refraction, without as well as with cycloplegia, the little help which the discussion of this paper may give to the optometrist may be far outweighed by the help which it may give us. There is every reason why the eye physician can and should do better noncycloplegic refraction than is ever possible to the optometrist. But he will not do it without sincere effort, and without frequently checking his noncycloplegic methods by cycloplegia. And he will continue to lose dissatisfied patients to the optometrist until he learns to check his cycloplegic methods by efficient accommodative control by overcorrection, and to use good medical judgment as to the proper part of the total error to be immediately corrected. One other point must be emphasized. Refraction is interesting and easy, or difficult and tedious, largely according to how well we do it. If refraction is drudgery to us, our patients know it.

Estimation of the Total Error.—This paper consists of (A) a brief discussion of the control of the accommodation by fogging and by cyclodamic methods. (B) The story of how cyclodamia developed. (1) How its novel but necessary methods of obtaining the maximum accommodative relaxation possible, and (2) of measuring the error thereby revealed without lessening such relaxation, were suggested by the analysis of everyday observations. (3) The description of the method as practically applied, and (4) the writer's estimate of its reliability.

(A) Accommodative Control.—The interference of the accommodation makes the measurement of refractive errors quite inaccurate, except in old people, unless the measurement is made under a large degree of accommodative control. There are, essentially, only two ways of obtaining such control. One is paralysis of the ciliary muscle by a cycloplegic drug, which obtains absolute control of the accommodation by abolishing it. The other is overcorrection with convex lenses, which withdraws most of the visual stimulus to accommodative action, and encourages accommodative relaxation by allowing better vision only as relaxation occurs. The greater the relaxation, the greater is the control established, total relaxation being equivalent to complete control. Since the chief function of the ciliary muscle is to provide better vision by appropriate contraction and relaxation (most of which is involuntary), overcorrection usually induces relaxation but

does not force it. The control afforded by overcorrection is therefore passive, and, though often adequate, is more variable, less complete and less certainly obtained than is that of cycloplegia. The amount of control obtained is indicated to some extent by the amount of relaxation, so that whenever overcorrection does not induce good relaxation control is uncertain.

The amount of relaxation induced by overcorrection has several factors. One of these is the relaxability of the ciliary muscle, which is governed more by the amount of accommodation habitually used than by the total power of the accommodation. Thus in some young hyperopic individuals who do little close work, powerful ciliary muscles sometimes relax so easily and greatly under overcorrection as to reveal most or all of the total error; while in some overworked hyperopic presbyopic patients, whose total accommodative power is much less, overcorrection may induce little or no relaxation. Nonrelaxability of the accommodation therefore is often a better indication of the need of a cycloplegic than is the age of the patient. Other factors in the amount of relaxation induced are the exact ways in which the overcorrection is applied, the amount of relaxation really sought, and the means used to measure the refractive error. Though the principle of overcorrection is the basis of all noncycloplegic refraction which attempts to control accommodation, various applications of this principle have resulted in several methods. Most of these methods, of which simple fogging is typical, attempt to obtain only so much relaxation as will reveal and measure that part of the total error which is to be corrected by glasses. This is done by the strongest convex or weakest concave lenses with which *maximum vision* can be readily obtained, no more accommodative relaxation being secured than the amount which maximum vision easily permits. The cyclodamic method, in sharp distinction to the others, seeks all the relaxation possible, and attempts to eliminate the effect of the accommodation altogether, so as to reveal and measure the total error itself. This is done by minute attention to detail in the arrangement of all the conditions of overcorrection so as to encourage continuous and cumulative relaxation to the point where relaxation is nearly or quite total. When such an amount of relaxation is not incompatible with sharp vision, the error may be measured as in fogging, but this is always uncertain. The error, therefore, has to be measured by the strongest convex or weakest concave lenses with which the *maximum re-*

laxation obtainable is observed, either during such skiascopy with distant fixation or during such subjective tests under blurred vision as afford no stimulus to accommodative action. Only in those cases in which the maximum relaxation observable is found or confirmed by fogging can we be sure that sharp distant vision has not excited any accommodative action whatsoever. Since the measurement is made at the instant of maximum relaxation, whenever that occurs, it is the closest approximation to the total error obtainable without a cycloplegic.

While all refraction methods give much the same measurement when the accommodation is very weak or absent, and all except cycloplegia are unreliable in the presence of real accommodative spacticity, they obtain widely different amounts of control when the accommodation is strong and active, sluggish in relaxation, weakly rigid, or stimulated by sharp vision, and thus reveal widely different amounts of error. Some of these amounts are definite enough to have received names. (a) The refractive error measured without any accommodative control whatever is called the Manifest or Apparent Error. This is so variable and uncertain a part of the total real error that it is measured only because the amount by which it differs from the total error affords information about accommodative behavior which is often needed in ordering correction. (b) The error revealed and measured by the methods of the fogging class is called the Full Manifest, or Apparent Error. This is usually about the amount which should be corrected, but is not dependably so in young people unless its relation to the real error is known. (c) The refractive error revealed when cyclodamic methods are used to bring about the maximum accommodative relaxation which can be measured by sharp vision may be called the Maximum Apparent Error. This is often considerably greater than the full apparent error revealed by simple fogging, and is close to the real error in so many cases that it may be used as a basis for determining very accurate practical correction, except in cases where considerable habit accommodation is inseparably associated with sharp vision, which are quite common. Such cases cannot be recognized by sharp vision tests. (d) The error revealed and measured by methods of the cyclodamic class, where the greatest relaxation obtained under skiascopy, blurred vision or fogging is the standard of measurement, may be called the Approximate Total, or Real Error. (e) The refractive error measured under cycloplegia is

called the Static Error. This differs from the total real error of the normal eye by the amount of error which is corrected by the minimal tonus of the ciliary muscle. Since measurement under cycloplegia is the only measurement which is always exactly the same under all conditions, it is the standard by which the accuracy of all other methods is gauged. It is quite undependable, however, unless cycloplegic paralysis is known to be absolutely complete.

Entirely dependable refraction requires measurement of the total or real error, of the full apparent error and of the manifest error. Three distinct types of examination, under absolutely different conditions of the accommodation, are necessary to obtain these measurements. The first type of examination measures the real error while the effect of the accommodation is eliminated, either totally by cycloplegia, or approximately by cyclodamia when it obtains good accommodative relaxation. The second type of examination determines the strongest lenses which do not blur sharp vision, and thus requires normal accommodative activity under the more moderate restraint which fogging affords. This examination is called postcycloplegic or postcyclodamic when it follows a first type examination, but is always essentially fogging in principle. The third type examination requires the presence of normal accommodative activity, either uncontrolled or without irksome restraint. It consists of the fine subjective tests which measure the manifest error and the visual acuity, and which often determine the exact cylindrical correction. These examinations will hereafter be called first type, second type, etc., when referred to in this paper. They are best made in the order mentioned, so that knowledge of the real error may guide the examinations in which accommodative behavior is studied.

(B) How Cyclodamia Developed.—There are so many young people whom, for various reasons, one would like to refract without a cycloplegic that if some method of eliminating accommodative interference and approximating the total error dependably could be found it would be very useful. The first hint that such a method was possible came to the writer during the draft examinations of the world war, when more dependence had to be placed upon objective than upon subjective methods of refraction, and unusual subjective checks which the draftee could not anticipate and be coached for had to be devised, because of occasional malingering with the hope of entering or evading service

unfairly. The observation that a person with myopia of 1.50 D. usually sees about 6/60 suggested tests under blurred vision, some of which proved useful. By combining these tests with distant fixation skiascopy, a crude but rapid method of roughly estimating the total error was worked out and used in several thousand cases for checking the visual acuity which the total error should allow against the visual acuity claimed. The observation was made during this unusual experience that, when cycloplegia was not used, the total refractive error could often be estimated more dependably by skiascopy checked by blurred vision than by sharp vision alone. This was the germ of cyclodamia.

Evidence confirming this observation was later afforded by a study of the difference between the amount of error revealed by the refraction method known as "Combined Skiascopy and Fogging" and that revealed by a subsequent cycloplegic. This method begins with skiascopy with distant fixation through lenses in a trial frame over both eyes, and goes over to fogging while the accommodation is still relaxed from the skiascopy, starting the fogging under the skiascopic lenses without removing the correction for the working distance. In the writer's use of this method, the lenses are then weakened equally until maximum vision is obtained, before testing either eye separately. The error revealed by this method was shown by cycloplegic check to be close to the total error when good skiascopic relaxation occurred and was maintained through the fogging. In many cases, however, the full skiascopic relaxation was not maintained, and the strongest lens with which the patient obtained maximum vision was weaker than that indicated by the skiascopy, sometimes much weaker. In these cases cycloplegia showed that the skiascopy was more accurate than the fogging, that the greatest amount of relaxation observed during the skiascopy was quite often practically total relaxation, and that unless the fogging confirmed the maximum skiascopic relaxation it did not reveal the approximate total error. The inferences from this study were: (a) That in some people there is so close a habit association between sharp vision and use of the accommodation that their complete disassociation is impossible without a cycloplegic. (b) That sharp vision actually stimulates accommodation in such people and so makes full relaxation impossible. (c) That the chief reason why full skiascopic relaxation in such people is not maintained throughout the fogging is the employment of sharp vision as the criterion of meas-

urement. (d) That sharp vision cannot, therefore, be used in such people for the estimation of the total error, unless the accommodation is paralyzed; and, conversely, that subjective measurement of the total error is impossible in such people without cycloplegia, unless it can be made without the use of sharp vision.

These inferences, together with the draft experience, suggested experiment as to the possibility of measuring refractive errors subjectively by such degrees of blurred vision as afford no stimulus to accommodative action. It was found that the great accommodative relaxation so often observed during skiascopy with distant fixation could not only be almost always fully maintained, and sometimes even increased, so long as the vision of both eyes was kept blurred to about 6/60, but that, if the astigmatism was previously corrected, the spherical error revealed by such relaxation could be quite accurately measured by deducting 1.50 D. from such overcorrection as blurred the vision to just 6/60. It was also found that the limit of accommodative relaxation was much more frequently reached when sharp vision was not allowed at all, and that the error revealed by this limit was not the Full Apparent Error revealed by other noncycloplegic methods but was a reliable approximation to the Total Error whenever relaxation was good.

A first type, noncycloplegic method of refraction, based upon these studies, was thus developed by the writer, and has now been used routinely for several years. This method was described in the American Journal of Ophthalmology for December, 1926, as "Refraction Under Cyclodamia." Cyclodamia literally means well controlled accommodation, the term being derived from the Greek *kuklos*, which has come to stand for the ciliary muscle, and *damnao*, which is the word the old Greeks used for the taming and control of their wild horses. Since noncycloplegic control of accommodation is only obtained when accommodative relaxation is induced by overcorrection, well controlled accommodation means accommodation at the point of the maximum relaxation obtainable. The terms "cyclodamia" and "maximum accommodative relaxation" may therefore be used interchangeably in their strict sense, "Cyclodamia" is also frequently used in a more general way to mean the "method of obtaining maximum accommodative relaxation and of measuring the error thereby revealed." Its usage, as well as its derivation, thus follows the pattern of "cycloplegia," which literally means paralyzed accommodation.

but is used in a general way to mean also the method of obtaining such paralysis and of measuring the error it reveals. In the 1926 description, the fact that the maximum accommodative relaxation obtainable was frequently greater than could be maintained when sharp vision was allowed, and that the error which it *then* revealed could *only* be measured by skiascopy and by blurred vision was not made clear enough to be understood. Cyclodamia was therefore confused with meticulous fogging. Cyclodamic control of the accommodation, however, is not limited by sharp vision, and it therefore obtains total or nearly total relaxation in many more cases than does even meticulous fogging. Total relaxation theoretically reveals the total error exactly, since it is less than the relaxation of cycloplegic paralysis only by the normal tonus of the ciliary muscle.

Maximum accommodative relaxation may be defined, for the purposes of this paper, as the largest amount of accommodative relaxation that can possibly be induced, during a comparatively short examination, by the continuous action of really favorable conditions. That is, it is the limit of obtainable relaxation. Since maximum accommodative relaxation is always required if the total error is to be approximated closely, we must consider: (1) The conditions under which such relaxation most surely occurs. (2) The methods of measuring the refractive error revealed by such relaxation which the maintenance of those conditions makes necessary. (3) The simplest practical way of securing those conditions and applying those methods in the examination of an individual patient. (4) The conditions under which this measurement is dependable.

(1). CONDITIONS UNDER WHICH MAXIMUM ACCOMMODATIVE RELAXATION MOST SURELY OCCURS.

Maximum relaxation is no more a natural state of the eyes, unless perhaps during sleep and in high myopia, than is cycloplegia, and it does not come by itself in a refraction examination but has to be carefully induced. While more or less relaxation is often easily obtained by ordinary fogging, maximum relaxation is only regularly secured by arrangement of all the minute details of the examination in such a way as to provide the most favorable conditions for it to occur. Many of these conditions were suggested by common observations.

One such observation is that if the strongest plus sphere with which a hyperopic patient gets maximum vision is determined

for each eye while the other is covered by a screen, he will usually get maximum vision with somewhat stronger spheres when both eyes are tested together. This suggests that when maximum relaxation is sought the eyes should always be tested together, under overcorrecting spheres at all times; that no screen be used, except for an instant when it is necessary to determine which eye sees the better at that moment; and that when one eye has to be excluded for a longer time it should be excluded by the addition of a still stronger sphere instead of by a cover or screen.

It is another common observation that when skiascopy shows that lenses in the trial frame over both eyes just cause reversal while the patient looks at the examiner, they will be too weak when the patient looks at a distant object, unless the accommodation is paralyzed. Maximum relaxation therefore requires that skiascopy be done with a distant point of fixation, just as in combined skiascopy and fogging, and that lenses in a trial frame over both eyes be used, instead of lens batteries which leave the other eye without overcorrection.

It is also observed that when skiascopy under such conditions is somewhat prolonged after reversal has been obtained, further, marked relaxation not infrequently occurs. Also that when one is called away for a moment from a patient who is wearing overcorrecting lenses which allow him about 6/60 vision in each eye, upon return his vision through those lenses will often be much better; though if he has removed the lenses, when they are replaced he may not be able to see even the 6/60 that he saw before. This suggests that maximum relaxation is favored by the continuous action of overcorrecting lenses over both eyes.

It is well known, though its great significance is not generally appreciated, that a patient with myopia of 1.50, if not allowed to improve vision by lid action, usually sees about 6/60. Also that if plus 1.50 sphere be added to any full cycloplegic correction, after the effect of the cycloplegic is entirely over, it will reduce vision to about 6/60, though it requires plus 2.25 addition to do this while the effect of the cycloplegic is complete, the 0.75 difference being due to the normal tonus of the ciliary muscle. The subtraction of plus 1.50 sphere from such overcorrection as allows a patient not under a cycloplegic just 6/60 vision should, therefore, be the measure of his spherical refractive error at that moment, and should also allow him normal vision if the amount of accommodation used remains the same at both the 6/60 and

the 6/6 levels. Experiment has shown that this is a quite accurate measure of his spherical refractive error at that moment, but that it allows normal vision regularly only in old people and quite irregularly in others. This is evidence that sharp distant vision often involves some involuntary accommodative action beyond the normal 0.75 tonus of the ciliary muscle, and so defeats maximum accommodative relaxation; that, when the latter is sought, sharp vision must be prevented; and that sharp vision cannot therefore be permitted in either eye, since accommodation in one eye causes accommodation in both.

Experiment has shown that maximum accommodative relaxation is most apt to occur either during skiascopy with distant fixation, or during such subjective tests as keep the patient looking with both eyes at distant objects or letters through lenses which blur the vision to an amount to which the patient is so unaccustomed that no stimulus to involuntary habit accommodation is provided, but an amount which does not entirely prevent some recognition. The exact amount of blurring most favorable to maximum accommodative relaxation varies somewhat in different patients, but is most often found at about the 6/60 level of blurred vision for both eyes or between that and the 6/12 level. When maximum accommodative relaxation is once obtained, it usually persists at both the 6/60 and the 6/12 levels, in some cases even at the 6/6 level, though it is seldom originally obtained there in young people. It is wise, however, not to permit vision better than 6/12 until the first type examination is over, since slightly blurred vision, such as from 6/6 to 6/10, does stimulate accommodation at times, especially in poor light. A similar condition is the slight blurring of full correction at the movies. Blurred vision at higher, unaccustomed levels does not ordinarily stimulate accommodation at all but tends to promote relaxation. The only exception to this which has been noted is that in a few people whom such blurring annoys, relaxation may be a little hard to start, and requires good light, a little time, and perhaps a little less blurring, so that some letters are sufficiently easy to recognize for the patient to make the effort to recognize them which he would not make before. Once relaxation begins, however, blurring regularly promotes it.

In such ways the conditions which most favor maximum accommodative relaxation have been worked out. They may be briefly summarized as excluding all tests which tend to stimulate

accommodative action, especially all subjective tests where the criterion is sharp vision, even though those tests do not stimulate accommodative action in all people. They exclude all tests of one eye alone, at any level of vision, unless the other eye is excluded by a greater amount of overcorrection instead of by a screen. They require uninterrupted overcorrection of both eyes of definite and measurable amounts, both during skiascopy with distant fixation, and during all subjective tests. And they require arrangement of all conditions of all tests so as to favor continuous and cumulative relaxation.

(III). METHODS OF MEASUREMENT NECESSARY UNDER MAXIMUM ACCOMMODATIVE RELAXATION.

The maintenance of the conditions under which maximum accommodative relaxation occurs determines what methods of measuring the refractive error which it reveals may be used and what may not. Among objective methods we are practically limited to skiascopy with distant fixation for measurement of both spherical and cylindrical errors. Accurate keratometry, however, gives an approximation to the cylindrical error in both axis and amount which greatly simplifies and shortens the skiascopic examination and makes it more readily exact, or it indicates that there is little or no astigmatism which is equally helpful. An extremely brief ophthalmoscopy, using minimum illumination, also gives a rough but useful hint as to the spherical error, and as to any disturbances in the mediae or retina which may interfere with measurement. Both should be done, however, before accommodative relaxation is sought, so that once started, its continuity may not be interrupted.

The same conditions likewise limit our subjective methods of measuring the spherical error to those which demand visual tests only at levels of blurred vision which do not ordinarily excite habitual accommodative action, such as those at 6/60 and at 6/12 and sometimes between. No subjective method for measuring the cylindrical error under blurred vision has been devised.

Since maximum accommodative relaxation occurs with any regularity only under overcorrection, and all measurements of the spherical error have to be made under that overcorrection, the amount of error which such relaxation reveals cannot be measured directly by the sphere under which it is observed, but always has to be measured by that sphere less a certain correction. If observed under skiascopy, the correction to be subtracted is, of

course, that for the working distance, viz., 1.D for 1 meter, 1.50 for 2/3 meter, 2.D for 1/2 meter, etc. If observed during subjective tests, the correction to be subtracted is that which blurs absolutely emmetropic vision to the same level as that at which the test was made, viz., 1.50 for 6/60 (20/200): 1.25 for 6/30 (20/100): 1.D for 6/20 (20/66): 0.75 for 6/15 (20/50): 0.50 for 6/12 (20/40). These corrections agree exactly with Thorington's table for vision under cycloplegia, if the normal tonus of the ciliary muscle is taken as 0.75, and have proven as accurate as the correction for the working distance in skiascopy. The idea of a correction to be applied in subjective tests is less familiar than is that of a correction in objective tests, though quite as logical. It is apparently the only way by which sharp vision, with its stimulus to accommodation, can be replaced in subjective measurement.

The methods of measuring the cylindrical or astigmatic error require more detailed description. Under cycloplegia both the cylindrical and the spherical error are constants, i. e., they are not variable and do not change in amount while full cycloplegia lasts. Without a cycloplegic the spherical error is always a variable, one limit of which we are trying to measure. The cylindrical error, however, may be considered as a constant, though there are occasional important exceptions. It is always much easier to measure the limit of variability in the sphere if we first eliminate the astigmatic constant. We therefore measure the cylindrical error first, so that we may put the correction for it into the trial frame, and thereby convert our patient's refractive errors into simple hyperopia or myopia while maximum accommodative relaxation is sought. Considerable accuracy in this measurement is necessary in both amount and axis, in order that the cylindrical correction may not leave enough uncorrected astigmatism to disturb the measurement of the spherical error. In finding this correction, we cannot, of course, use the fine, exact subjective tests with charts, dials, trial lenses and cross cylinders which are so useful under cycloplegia and in third type examinations, since all of them tend to stimulate accommodative action. We have, therefore, to be content with only objective measurement of the astigmatic error at this stage, though as accurate as we can make it, and postpone all subjective measurement to the second and third type examinations. Such objective measurement is perhaps most exactly made by first accurately measuring the corneal astigmatism with a good keratometer, such as

the 1920 Meyrowitz model: then putting the cylinders thereby indicated into the trial frame, together with overstrong spheres, and accurately skiascoping, first finding the spheres which bring reversal in the weaker meridian, then changing the cylinders from the correction indicated by the keratometer so far as is necessary to correct the total astigmatism shown by skiascopy. Since the corneal astigmatism is almost always much the largest factor in the total astigmatism, and very often the only one, such changes are usually quite small. This objective correction of the total astigmatism, when later checked by the fine subjective tests of the third type examination, is usually found to be a very close correction, and not infrequently closer to that found under a subsequent cycloplegia (since it is made under more nearly the same conditions), than is the correction indicated by second and third type examinations alone. This objective correction is therefore left in the trial frame without further change or attention during all the efforts of the first type examination to obtain maximum accommodative relaxation.

(III). THE REFRACTION OF A PATIENT UNDER MAXIMUM ACCOMMODATIVE RELAXATION.

While the history may be taken by an assistant, no preliminary test of vision or refraction which may possibly stimulate accommodative action should precede the first type examination. The latter begins with an inquiry by the examiner as to the patient's chief symptoms and their relation to use of the eyes and to general health. Any other necessary facts will be volunteered by the patient or will be suggested by the behavior of the accommodation during the examination. An accurate keratometer reading by the examiner, a very brief ophthalmoscopic examination, and a record of the glasses worn, complete the preliminary data. The total astigmatism is then determined as a variation from the corneal astigmatism by skiascopy with distant fixation, as previously described, and we are ready to go ahead with the induction of maximum accommodative relaxation and the measurement of the spherical error which it reveals. This is done in a short but continuous examination with three records.

Both eyes alternately are further skiascoped under the cylindrical correction found, together with overstrong spheres in the trial frame, always with a distant object of fixation in a moderately dark room, until as much accommodative relaxation as

possible has been obtained: not only gradual relaxation, but also, if possible, that sudden, greater relaxation which sometimes lasts but a moment. The skiascopy should not be hurried, as a little time for relaxation to occur has to be allowed, but it should not be prolonged unduly. The spheres are meanwhile changed to those which just cause reversal at the moment of greatest relaxation, regardless of when that occurs or whether it is maintained or not. The lens combination then in the trial frame, less correction for the working distance, exactly measures the total error thus far uncovered, and this amount should be recorded as the skiascopic measurement. It saves a change of lenses for the next test, if skiascopy has been done at 2/3 meter distance, for which the correction is + 1.50 D.

Without removing the skiascopic lenses, and so giving the relaxed accommodation a chance to become active again, subjective tests of both eyes together are begun, beginning with the lenses then in the trial frame. Since these are + 1.50 overcorrection of the total error found at the moment of greatest relaxation under skiascopy, if this relaxation is maintained the patient should see just 6/60 with them, and this is what is usually observed. In some patients this relaxation is not maintained and they will not see quite 6/60 at first. And in a few patients more relaxation occurs and they will see better than 6/60, sometimes much better. If the patient does not see 6/60, a little time for relaxation should be allowed. Many patients say that everything is so blurred that they can see nothing, and make no effort to see. But if gently encouraged, told to look away and look back, asked to tell what they think or guess what the big letter is in spite of the blur, they will usually name it correctly. This is sufficient, if they have not seen it before so as to remember it, and if they are unable to name those beneath it. If it becomes evident that the 6/60 letter cannot be named correctly the spheres over both eyes are weakened slightly, each by the same amount, until it is just surely recognizable. Since both eyes so far have been tested together, we do not yet know whether the patient sees 6/60 with both eyes or with only one. This fact is now determined by covering each eye alternately with a card for an instant: but only for an instant or the accommodation may tighten up and he may not see 6/60 with either eye. If he has seen it with only one eye, that eye is excluded by the addition of a slightly stronger sphere (+ 0.50

to + 1.D), while the vision of the other eye is brought to 6/60 by a slight change of sphere. If the patient sees better than 6/60, either at first or at any time later, the spheres are increased until each eye sees just 6/60. This practically amounts to fogging of both eyes together to 6/60 instead of to 6/6. In changing spheres, one should be put into the trial frame before the other is taken out, as neither eye must be allowed to see at all without overcorrection, even for the instant of changing lenses, during the whole first type examination. The 6/60 letter must also be well illuminated, as accommodation tends to tighten up in poor light. The 6/60 test is very simple and seldom takes more than two or three minutes unless accommodation is very refractory or the patient does not cooperate. It may be briefly summarized as follows: Starting with the skiascopic lenses, 6/60 vision is expected and usually obtained. If vision is not just 6/60 after a minute or two of encouragement, the spheres are changed equally until the 6/60 letter is just surely recognized and no more: first for the two eyes together, then for each eye separately while the other is excluded by a stronger lens. When all the relaxation possible has been obtained and each eye sees just 6/60, the lenses then in the trial frame, less + 1.50 D., measure the total error with 6/60 vision, and this should be recorded as the 6/60 measurement.

A similar test at the 6/12 level of blurred vision completes the first type examination. This ordinarily requires lenses just 1.D. weaker than those of the 6/60 test, and so takes very little time. In some cases, however, especially those where good cooperation in the 6/60 test was not obtained, and those in which fatigue is at the point where it aids relaxation and has not reached the point where it defeats it, more relaxation is found at the 6/12 level than at any time before. In any case, the lenses which permit 6/12 vision and no more, less +0.50, are the measure of the total error uncovered by 6/12 vision, and are recorded as the 6/12 measurement.

Comparison of the skiascopic, the 6/60 and the 6/12 records completes the first type examination. In most cases these records are practically the same. If they are not, relaxation has not reached to the same point in each of the three tests. Accommodation must therefore have been used in some of them, perhaps in all, and the approximation of the total error is uncertain. Reexamination under better conditions or recourse to

cycloplegia is therefore needed. If neither of these are practicable, that one of the three records which shows the largest amount of error is taken as the closest approximation to the real error so far made. When the three records agree, accommodative relaxation must have reached to the same point in all of the tests. It is probable that this point is total relaxation, or very close to it, since any relaxation less than total should be variable, unless there is tonic spasm or rigidity of the accommodation which remains absolutely fixed in amount during the varying conditions of the three tests. The latter is extremely improbable when any real amount of relaxation has been obtained. There is also less reason why relaxation, once well started and not limited by the habits of sharp vision, should not go on to total relaxation under favorable conditions than stop at any point short of that. The agreeing records, when relaxation is good, are therefore considered as a reliable approximation to the total real error. This is then used as a guide in the second and third type examinations just as if it has been made under cycloplegia, with the exception that it must always be less than the static error by the 0.75 average tonus of the ciliary muscle, and by so much more as the maximum relaxation observed may be less than total relaxation, which is usually very little unless relaxation has been poor.

Throughout this first type examination, which usually takes five to ten minutes when one is familiar with it, it must never be forgotten that the amount of spherical error revealed varies with the amount of accommodative relaxation, and that we are interested only in the greatest amount of error observed at any time. We therefore do everything possible to promote relaxation, and watch for the instant at which it is greatest in each of the three tests. The greatest amount of error surely observed, though but for an instant, should not therefore be distrusted. Nor should it be forgotten, during this first type examination, that we are not at all concerned with the amount of error to be corrected. That will be determined later, as a variation from the total error, by the finer subjective tests of the postcyclodamic examination. It is the total error alone which we are here trying to approximate reliably, and so avoid the necessity of a cycloplegic. Much confusion results if this is not kept clearly in mind. The cyclodamic examination, therefore, can be and is largely objective, and demands from the patient only: (a) Dis-

tant fixation during skiascopy; (b) The reading of 6/60 and 6/12 correctly and of 6/30 and 6/10 incorrectly with eyes wide open; and (c) The information as to which eye sees the better when that needs to be known. Since no sharp vision and no choice of lenses is permitted, the cyclodamic examination is essentially a rapid one.

While first type cyclodamic examinations seeking the total error, and the ordinary noncycloplegic examinations seeking the amount of correction to be ordered, have to be made separately and cannot be combined, both because their objectives are different and because they require entirely different states of the accommodation, there should be no interval between the two examinations. Measurement under blurred vision is seldom understood by the patient, and he is often rather disappointed until he is allowed maximum vision and the choice between lenses of the second and third type examinations. It is therefore usually wise, as soon as the 6/12 measurement is recorded, to proceed with the second type examination at once by removing the 0.50 overcorrection of the 6/12 level and seeing if maximum relaxation can be maintained with maximum vision, as it often can when habit accommodation is not too strong. The usual well known methods of second and third type examinations are then used. The spheres are first weakened, if necessary, until maximum visual acuity is obtained. The cylinders are adjusted by fine subjective tests in which cross cylinders are very useful; the manifest error and the visual acuity with and without correction determined and recorded; the spheres again strengthened as much as maximum vision surely permits; and the final correction balanced, if necessary, to give each eye equal conditions of vision. The combination of lenses then in the trial frame is usually the prescription for glasses; unless it does not correct enough of the total error to give almost certain relief from symptoms, or corrects so much more than the full manifest error as probably to remain uncomfortable for the patient beyond the ordinary adjustment period of two to four weeks. The probable effect of any change from the previous correction upon the relation between accommodation and convergence, and upon temporary or permanent phorias, must also be considered. When the amount of previously uncorrected spherical error in young people is large, it is usually better to correct it by degrees than to try to force full correction at once, unless there is urgent need for full correction.

(IV). THE RELIABILITY OF CYCLODAMIA IN APPROXIMATING THE TOTAL ERROR.

Unless fogging reveals considerable previously uncorrected error, it is not a dependable guide to adequate correction in young people and in overworked presbyopic individuals. Knowledge of the total error is necessary for real dependability. On the other hand, the total error found under cycloplegia or cyclodamia is not, by itself, a dependable guide to satisfactory correction, except in myopic patients. The right amount of concession to the accommodative habit of a patient can only be found in a postcycloplegic or postcyclodamic fogging examination of that patient, where knowledge of the total error keeps the concession from being more than is surely necessary for ultimate comfort. The right amount is too variable in different patients to be determined by arbitrary rules or upon the basis of a precycloplegic examination. Since the total error, therefore, is seldom fully corrected, a reliable approximation to it is all that is usually needed for dependability. The method described in this paper affords such an approximation whenever the accommodation is mobile. While the method is, of course, by no means as generally dependable as is thorough atropin cycloplegia, it falls little short of that in the cases where it obtains more relaxation than can be measured by sharp vision. It is more dependable than homatropin cycloplegia as that is perhaps most often used. It is much more dependable than fogging, since it approximates the total error reliably in very many more cases, and since it points out, by failure to obtain relaxation, almost all of the hyperopic cases in which its reliability is uncertain. Much that is obviously impossible with other noncycloplegic methods is thus practicable with cyclodamia; how much we do not yet know. The only accommodative anomaly affecting distant vision which it does not make evident, and in the presence of which it is therefore most apt to be unreliable, is tonic accommodative spasticity. This, in its very rare active form, is easily recognized from the symptoms. Its passive form is seldom seen except in overworked eyes, where it is essentially a habit accommodation associated with sharp vision which has become so fixed that it will not relax even under the blurred vision of cyclodamia. This form can only be inferred from the failure to get good relaxation, together with the symptoms and the history. Ordinary habit accommodation, unrelaxable under sharp vision but relaxable under blurred

vision, and clonic accommodative spasticity, both of which are much more common than tonic spasticity, do not interfere with cyclodamic control. The writer, after several years of experience with the cyclodamic method, checked by atropin cycloplegia in many cases, has noted practically no exceptions to its reliability in closely approximating the total error whenever relaxation was good and all its tests agreed. When relaxation was poor, however, when its tests did not agree, and when it revealed no more error than did fogging, cycloplegic check showed that it was often accurate, but not reliably so.

The behavior of the accommodation during a cyclodamic examination usually shows whether a cycloplegic is, or is not, needed to reveal the total error, as well as affords other valuable information. For these reasons routine cyclodamia, with cycloplegia whenever indicated, has proved more satisfactory to the writer than routine dependence upon either. Cyclodamia may, of course, be used as an alternative method when cycloplegia is not desirable. A better way, however, is to use it routinely instead of a preliminary manifest, since it takes little more time and gives much more definite information; and its sureness, smoothness and rapidity improve greatly with routine use. When its approximation to the total error shows the earmarks of reliability, it should be followed by a balanced fogging for the determination of the correction to be prescribed, just as after cycloplegia. When its reliability seems in any way doubtful, it should be followed by a cycloplegic. As one learns the possibilities and the limitations of the method by experience, watches its revelation of the anomalies of the accommodation, which are so well described in Duke-Elder's "Practice of Refraction" that the chapter there devoted to them should be read by every refractionist, and comes to know the amounts of relaxation to be expected with different kinds and degrees of refractive error, he will find that he needs a cycloplegic much less often.

In the method of estimating the total error and in the theory of cyclodamic control here described, the prevention of all sharp vision with either eye, the subordination of all else to the obtaining of continuous and cumulative accommodative relaxation, the measurement of the error only at the utmost limit of relaxation, the subjective tests under blurred vision with correction for the amount of blurring, and their arrangement (together with the skiascopic part of combined skiascopy and fogging)

into a definite method, seem comparatively new, since they have heretofore received little attention. On the other hand, all of the facts upon which are based the unusual methods of measurement necessary if full cyclodamic control is not to be defeated, may be found in the textbooks and in the literature.

SUMMARY.

Knowledge of the accommodative habit and behavior of every patient is essential to the determination of the proper correction of his refractive error. This knowledge is only obtained by comparison of the total error with that part of the error revealed by the fogging and manifest examinations. While the total error may always be measured under cycloplegia, and in some cases only under cycloplegia, it may often be sufficiently closely approximated by the cyclodamic method described in this paper: quite dependably so when good relaxation is obtained and all its tests agree; less dependably so in other cases. This method obtains an amount of accommodative control which is usually greater and always more reliable than that of fogging, and often but slightly less than that of cycloplegia, by making every smallest detail of the examination contribute to the production of maximum accommodative relaxation. The refractive error thereby revealed often has to be measured without the use of sharp vision, which has been shown to prevent maximum relaxation in many young people. Once the novel principles and methods of cyclodamia are well understood, it is easier and quicker than cycloplegia, and may well replace it in young people as well as old, if recourse is had to cycloplegia whenever relaxation is poor or accommodative behavior is irregular. Since it differs from all other noncycloplegic methods in that it is the total error which it reveals or approximates when good relaxation is obtained, it should always be followed by a fogging and manifest examination, and the correction determined as a variation from the total error, just as after a cycloplegic.

DISCUSSION.

DR. S. J. BEACH, Portland, Maine: As a more reliable substitute for cycloplegia than fogging and the like, cyclodamia has an obvious field. This is true where the atropine group is contraindicated, and in the larger group where its use is inexpedient. Take the recent case of a college student seized with acute eye-strain in the midst of examinations, or a banker arranging the details of a merger.

What I really wish to insist upon is the value of this method as a check upon cycloplegic refraction. We are too prone to forget the only moderate reliability of atropine, not to say that the relaxation from homatropine resembles complete cycloplegia only as mist resembles rain. In cases where it is desirable to know the total error of refraction—and this should include most of our work—I find Dr. Smith's method more trustworthy than much that passes for cycloplegia.

Dr. Edward Jackson, Denver: One who realizes the great assistance of cycloplegics and uses them whenever it is practicable, knows that their use is not the whole of the art of refraction. By reliance too exclusively on cycloplegics, as Dr. Beach has pointed out, the ophthalmologist misses his opportunity to demonstrate that he knows about refraction all that does the optometrist.

Another point with reference to measuring refraction without cycloplegics is the irregular astigmatism of the dilated pupil. There are many cases of irregular astigmatism with variations in the peripheral zone of the dilated pupil. In many cases that does not influence the results obtained with cycloplegia and the necessary mydriasis. But there are other cases in which the exploring of that peripheral zone is important. The patient uses his eyes to see at a distance outdoors in strong light, or to do near work, with contracted pupils. To think the size of the pupil has nothing to do with refraction, and to accept the cycloplegic showings on that basis, is a very serious departure from the effort which every ophthalmologist ought to make to find out all about the refraction of his patient and then to use his knowledge to the patient's advantage.

On page 52 of the Presession volume is a single sentence that I think should be omitted to bring about more confidence in and respect for cyclodamia. This sentence reads: "No subjective method for measuring the cylindrical error under blurred vision has been devised." We have demonstrated certainly in the last year to fifty ophthalmologists, in a way that convinced them, that the cross cylinder can be used so as to provide accurate information as to the amount of astigmatism, while the vision is purposely blurred. The blurring of vision depends upon the size of the pupil. That has been overlooked by Thorington and others. But with the blurring caused by one diopter spheric overcorrection, I have demonstrated to the doctor himself the proper strength and direction for the cylinder.

A change of 5° in the axis would enable him to pick out immediately which way the cylinder had departed from the proper axis. And one-quarter diopter of error in the strength of the cylinder could be equally well demonstrated.

Dr. W. B. Lancaster, Boston (Using slides): Dr. Jackson brought out one or two points that I wish to emphasize. One is the difference in refraction in different parts of the cornea—often a difference of more than one diopter.

This figure shows the refractive power of different parts of the cornea of Tscherning's right eye. You see that near the center in one place it is E; in another M i; near the periphery we have such large differences

as M5, M2, M3.5, H 1. (The figures are diopters. Thus, M 1 means myopia of 1 diopter.) The differences in refraction in the visual part of the cornea are not so large; that is what the pupil is for. Enlarge the pupil, and errors are bound to creep in from this obvious source.

For my part, I find I get much more accurate measurement of refraction when I do not use a cycloplegic. I advise everybody to observe this rule: do not do refraction *without* a cycloplegic until you have learned to do it better that way than any other. You owe it to your patient to follow the method by which you get the best results. If you try to measure the refraction without a cycloplegic following the *same technique as with*, you certainly will not succeed. You have to do as Dr. Smith has done—study the science and art of refraction without cycloplegia, before you can expect to do it successfully.

Another point I wish to emphasize is the amount of reduction of vision from the addition of different spheres. For many years I have made a record in each case of refraction, not only of the visual acuity with the correction, but the visual acuity with a + 50 added. It is a useful guide. If the + 50 does not reduce his vision one or two lines, I know that I am giving the patient an undercorrection. There may be a reason for doing so, and this little notation makes it clear in the record. The chart I use is so devised (Ewing after Green) that the difference between any two consecutive lines is the same throughout the chart; each line is the same percentage larger than the one below it. So if I reduce the visual acuity one line anywhere on the chart, it takes the same amount of blurring of the vision to do it. I find that the + 50 on my chart reduces about two lines—not as much as Dr. Smith finds it. The reason for differences of opinion on this is chiefly a matter of the size of the pupil. It is perfectly obvious, if you make a little drawing of the cone of rays that come from the pupil to the retina, that the amount of blurring caused by moving the focus farther from the retina forward or back by a plus or minus glass depends on the strength of the glass and also on the size of the pupil. But that is a minor point. The main point is that here is a man who has studied the method of measuring the patient's refraction and has got somewhere in doing it.

Dr. William H. Crisp, Denver: Dr. Lancaster's argument, based upon the drawings of Tscherning, assumes that the dilated pupil influences refraction in this way: The rather general impression is that when you dilate a pupil your patient at once begins to use the focusing effect of the whole area of the cornea and lens. In my opinion, there is no satisfactory reason for believing that to be the case. I believe that in a large majority of cases the patient still continues to select a preferred central area of the enlarged pupil. In other words, he does not use all of the peripheral area of the pupil. So if you base too much on this conception of the behavior of the eye you will sometimes sacrifice the value that may come from cycloplegia.

One of the greatest sins in refraction is carelessness as to precycloplegic estimation of refraction. At the precycloplegic test we obtain some information that cannot be obtained from a postcycloplegic estimation, and

with the great advantage of more accurate understanding of the case before the cycloplegic is used. Too many people think it impossible to do any refraction work whatever without a cycloplegic. I agree with Dr. Smith on that. I should say that in at least three cases out of four, if one makes a sufficiently deliberate precycloplegic estimation of the refraction, he will come very close indeed to the patient's correction. One reason examiners fail in working without a cycloplegic is that they are not sufficiently deliberate in applying the fogging method, first to each eye and then to both eyes together, giving the patient plenty of time for relaxation. There is an unfortunate lack of knowledge of the advantages of bilateral fogging, especially after one has worked out the measurement for the individual eye. Often one obtains much fuller relaxation of the hyperopic eye on fogging both eyes together after the astigmatism has been worked out for the individual eye. In the last year or two I have been using contralateral fogging in tests with the cross cylinder. But in the majority of cases the single eye is not most satisfactorily worked on as to cylinder by keeping it decidedly fogged. Usually one obtains a decidedly more accurate astigmatic measurement by having the accommodation of the eye under test barely relaxed so that the patient is not using his accommodation but is getting his best visual acuity and is not overcorrected by the plus sphere.

I think we should all pay more attention to careful refraction without a cycloplegic, even though we use cycloplegia in many cases.

Dr. Dorland Smith, closing: I wish to thank the gentlemen who have contributed to my information on this subject, particularly Dr. Jackson, for calling my attention to the fact that it is possible to measure the astigmatism under blurred vision.

SPOROTHRIX INFECTION OF THE EYE AND ADNEXA.

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Thirty-two proven cases of sporothrix infection of the eye and adnexa have been recorded in the literature. Of these only seven have been reported from this country, most of these originating from the Mississippi Valley. It is therefore obvious that this is a rare condition.

Infection occurs by invasion of the tissues by the sporotrichum, one of the fungi imperfecti. This organism was discovered by Schenck in 1898. A similar organism, but with cultural variations, reported later by Beurmann, is thought by some workers to be identical with the former.

Most any tissue may be involved, but it is usually primary in the skin. Gifford[1] says: "It most commonly is primary in the hand following a superficial abrasion. The lymphatics are nearly always involved."

Usually there is a subcutaneous nodule which breaks down to form an indurated ulcer resembling the primary lesion in lues. The French have reported several cases of systemic involvement with deep gummata. The infection is very apt to form discharging fistuli.

The tissue changes are not pathognomonic and diagnosis depends on isolating the organism. In a case which I shall report Dr. F. A. Verhoeff demonstrated the organism in the tissue by means of Verhoeff's modified gram stain. As sporothrix grows on glucose agar or Sabourard's media, but is difficult to find in the smear, cultural diagnosis is the method of choice.

The treatment generally employed is potassium iodide by mouth in large doses, iodine, gentian violet and Lugol's solution locally. S. R. Gifford[1] has tried injections of 7 per cent solution of potassium iodide in and about the lesions but did not continue because of the intensely painful reaction.

Potassium iodide has been considered almost a specific and cures have been repeatedly reported.

The prognosis is thought to be very good, and Gifford[1] writes that "a correct diagnosis once made is gratifying because potassium iodide in large doses nearly always brings about prompt recovery."

The first case of sporotrichosis of the eye and adnexa was reported in 1907, by Danlos and Blanc[2], and was one of lower lid infection. Morax and Carlotti[3] reported a lid case in 1908 which promptly healed under potassium iodide.

Fava[4] reported a laboratory infection of his own conjunctiva in 1909. There was marked involvement of the lymphatics, also an eosinophilia of 6 to 8 per cent. Cure was rapid with potassium iodide.

The same year Morax[5] cited a case of primary sporotrichosis of the conjunctiva cured by potassium iodide. There was marked involvement of the cervical lymphatics. The organism was identified by him as sporotrichosis beurmanni.

Burnier,[6] in 1909, reported a case with multiple subcutaneous nonulcerating nodules, on the lower lid of each eye. These were painless. There was enlargement of the preauricular gland. A lesion on the hand ulcerated and sporotrichosis beurmanni was isolated. The nodules were treated with galvanocautery. Later all lesions healed with large doses of potassium iodide. An eosinophilia of 6 per cent persisted after cure.

In 1909 Beurmann[7] and Gougerot reported a fatal case of general sporotrichosis with multiple gummata and abscesses with destruction of one eye.

The following year Jeansulme and Poulard[8] reported a generalized sporotrichosis with involvement of the iris. The case recovered under potassium iodide but the pupil of the involved eye remained obstructed with exudate, with only light perception.

The same year Morax[9] reported another case of abscess in the orbit, with fistula in the upper lid. The patient refused to take potassium iodide and the lesion did not heal while under observation. Sporotrichosis beurmanni was isolated.

The same year Velter[10] reported a case of orbital abscess which failed to improve when the patient refused to continue potassium iodide.

In 1910, H. Gifford[11] reported the first ocular sporotrichosis in the United States. The infection was in the conjunctiva and showed a nodular thickening. Sporothrix was isolated and the

author expressed doubt as to the difference between the Schenck and Beurmann types of organisms.

H. Gifford had seen five other cases of ocular infection which he is convinced were sporotrichosis but upon which the organism was not isolated, although cultures were taken.

Chaillous[12] reported a case in 1911 with infection of the conjunctiva from handling a horse that had suppurating eyes. Sporotrichosis beurmanni was isolated.

The same year Morax[13] reported a case of primary lacrimal sac infection with marked involvement of the lymphatics. The preauricular gland was enlarged and nodules appeared in the soft tissues of the cheek. These improved rapidly under potassium iodide. Sporotrichosis beurmanni was isolated.

Dorr,[14] in 1911, saw a case of corneal involvement in an infant in a generalized sporotrichosis with inflammation of the surrounding conjunctiva. All lesions healed with potassium iodide.

A case of systemic infection by sporothrix with involvement of the bones and gumma of the iris in one eye is reported by Legry, Sourdel and Velter[15] in 1911. It improved with potassium iodide.

In 1912, Lapersonne,[16] in reporting on this same case, stated that he isolated sporotrichosis beurmanni from pus taken from the interior of the involved eye, by means of a Luer syringe. He described another case of systemic infection with iridocyclitis which resulted in atrophy of the eyeball. The other lesions healed under potassium iodide, but the eye became painful and had to be removed. Sporotrichosis was found elsewhere but not in the eye tissues.

Chaillous[17] at this time reported a primary lid sporotrichosis with involvement of the eyeball and multiple subcutaneous nodules on the body with no enlargement of the lymphatics. The eye was lost, but the other lesions cleared with potassium iodide. Cultures were positive for sporothrix.

In 1913, Morax[18] described a cure of a sporothrix infection of the lower eyelid with potassium iodide.

The following year[19] he reported a case of uveitis due to sporothrix infection without other demonstrable lesions. Sporotrichosis beurmanni was cultured from pus drawn from a puncture in the eyeball.

Bedell,[20] in 1914, reported a case of sporothrix of the conjunctiva and lacrimal sac with a detailed description of the organism

cultured. Cure was obtained with potassium iodide and iodine locally.

Another case of sporotrichosis of the eye due to laboratory infection was reported by Wilder and McCullogh[21] in 1914. The infection was conjunctival with marked pain and swelling and enlargement of the lymphatics. There was fever and leucocytosis but no lesions elsewhere on the body. Recovery was prompt with potassium iodide and iodine.

Dwyer and Larkin[22] the same year reported a case of lid infection with the primary lesion on a finger.

In a case reported by Aloin and Vallia,[23] in 1920, there was involvement and perforation of the frontal bone along with an abscess of the upper eyelid. This case cleared rapidly with potassium iodide.

A case is reported from Egypt, in 1922, by Sobhy Bey[24] with infection about the inner canthus. Cultures showed different characteristics than those described by Schenck and Beurmann. However, this lesion healed with potassium iodide.

In 1922, S. R. Gifford[1] reported a case of lacrimal sac infection with formation of a fistula that extended back to the ethmoid cells. These were full of pus. Intranasal exenteration of the ethmoids and trichloracetic acid and iodine locally with potassium iodide internally brought about a cure. The sporotrichum was isolated.

S. R. Gifford[1] noted two other cases not proven by culture which he believed to be sporothrix, and both were cured by potassium iodide.

In 1924, S. R. Gifford[1] reported another case of lid infection from which sporothrix was isolated. In addition to potassium iodide internally a 7 per cent solution was injected about the lesion. Complete healing occurred.

King,[25] in 1927, reported a generalized sporotrichosis involving the eye, in which improvement was slow with potassium iodide until large doses were given intravenously. X-ray was also used. All lesions finally healed.

Alvis,[26] in 1928, described a case of chronic trachoma complicated by shallow ulcers from which he isolated sporothrix. Potassium iodide did not heal these lesions and local applications of iodine were of no avail. No further extension was reported.

Three years ago I reported a case of sporotrichosis primary in the upper lid, before the New England Ophthalmological Society. This case has shown many subsequent developments.

Case report: G. G., aged 28 years, of French-Canadian parentage, by occupation a telephone linesman, consulted me five years ago because of a small nodule on the upper right eyelid. It had been present, slowly increasing in size for about ten years. There were no other symptoms.

One year before the nodule had been opened and curretted, as a chalazion, by another physician. It enlarged more rapidly but showed no tendency to break down.

Examination showed an irregular almond shaped nodule, six millimeters in diameter, midway on the upper lid, over which the skin was slightly adherent. Findings were otherwise negative except for an enlarged cervical lymph node on this side, which the patient claimed to have been present without change since infancy.

Complete removal of the nodule was attempted, dissection being carried well around the mass. The diseased tissue was dense and fibrous.

Frozen section, as well as a prepared section, was diagnosed, "Giant Cell Sarcoma." Mallory reported, "Giant cell inflammatory tissue due to fungus growth," which Verhoeff identified as, probably, sporothrix.

Soon after operation a discharging fistula appeared at the site of the incision. Sporothrix was isolated by Verhoeff.

Potassium iodide was started in large doses, and, although well borne, the growth extended to the outer canthus with formation of several fistuli. During the following two years the infection progressed, slowly involving the tissues over the lower rim of the orbit. Several large nodules were removed surgically and the wounds cauterized with trichloracetic acid. Potassium iodide was used intermittantly to toleration. Roentgen-ray therapy was tried for several months without effect. Salvarsan had been used intravenously. Local treatment with iodine, gentian violet, and Lugol's solution, and local injections of gentian violet and mercurochrome were used. During this period enlarged lymph nodes had been removed from both the right side of the neck and the right inguinal region. These showed no organisms and only epithelioid infiltration.

Roentgenograms of the skull and lungs were negative. The blood gave a constant eosinophilia of 7 per cent.

For a period of six or eight months the patient was not under observation. When seen again there was a marked exophthalmos of the eyeball, and a large hypopion ulcer covering the lower half of the cornea. The chemosis and injection of the conjunctiva was extreme. Light perception was doubtful. There was a large indurated mass extending down from the lower rim of the orbit nearly the size of a hen's egg. For the past three weeks he had suffered his first pain, which was now intense.

Roentgen examination showed "partial destruction of the right malar bone and of the external angular process of the frontal bone with areas of rarefaction. Antrum cloudy."

Due to the apparent involvement of the paranasal sinuses Dr. F. T. Hill was called in consultation. Radical surgery seemed to offer the only hope of relief.

Under rectal anesthesia the orbital contents were exenterated along with a wide dissection to include all the diseased tissue. This included the outer wall of the orbit to the zygomatic arch and its floor, exposing the antrum. The outer wall of the antrum was removed. Dissection of the soft tissues was performed with surgical diathermy. A large skin flap was saved. The patient reacted nicely, and was free from pain following operation.

Three months later a nodule had to be removed from the canine fossa under local anesthesia. For five months there has been no sign of recurrence about the face, and the patient has been able to do about as he pleases. He now complains of a slight edema of the right leg after any length of time upon his feet. There is a slight deep induration in the inguinal region where the glands were removed.

At the present time the patient has been receiving postassium iodide intravenously.

Recent roentgenograms of skull, lungs, pelvis, and long bones showed no pathology.

The continuous progression of the infection in spite of repeated surgery and therapy with evident metastatic involvement of the inguinal glands gives rather a dubious prognosis.

Verhoeff's pathologic report on this case is as follows: "All the tissues submitted for examination, including the bone tissue, show the same inflammatory process, except the lymph gland.

The normal tissue has been pervaded with granulation tissue which has gone on to the formation of dense fibrous tissue. (Fig. 1.) The tissue contains large numbers of foreign body giant cells, but no tubercles resembling tuberculosis or syphilis. Some of the tissue shows considerable infiltration with lymphocytes; some, only moderate infiltration with these cells. By ordinary methods of staining no organisms can be found in the tissues, but Verhoeff's modified gram stain shows filamentous organisms scattered through the tissues, some of them of great length. (Fig. 2.) Some of them are surrounded by giant cells, others are not. The lymph gland shows the lymph sinuses distended with epitheloid

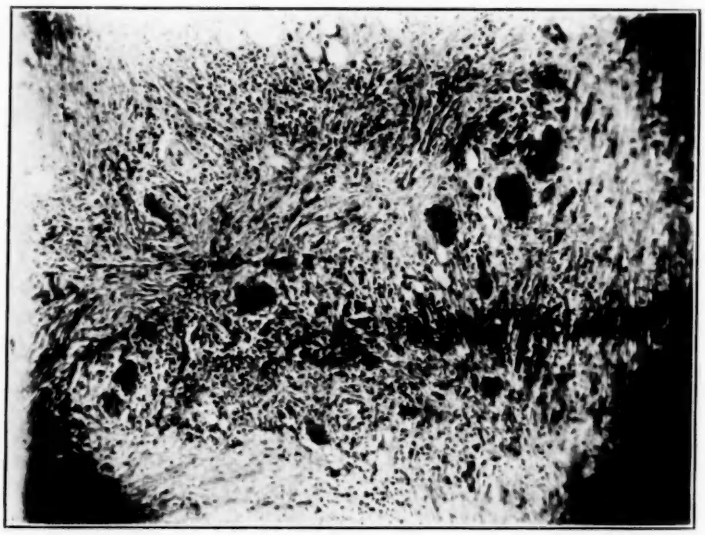

Fig. 1. Section of diseased tissue stained with Hematoxylin-Eosin.

cells. Smears from the pus from the lesion in the lids show sporothrix filaments in considerable numbers. A culture taken on glucose agar showed the organism in four days. Transplants from the original growth on glucose agar and Sabaraud's media failed to grow. Pieces of the original culture media were fixed in formalin, sectioned and stained in Verhoeff's modified gram stain. These show the organisms growing down into the medium in characteristic fashion." (Fig. 3.)

Therapy.—The treatment of sporotrichosis, it would seem, is not entirely perfected. Potassium iodide, although formerly con-

sidered almost a specific, does not always prove successful. In this case it has been forced to toleration, over a period of five years, with no beneficial effect.

Dr. F. T. Hill[27] suggests that the action of potassium iodide is probably the same as it is in tertiary lues, that of breaking down fibrosis, thus allowing the natural defenses of the body as well as those introduced therapeutically to act upon the organism, rather than any specific reaction for the sporotrichum inherent in the drug itself.

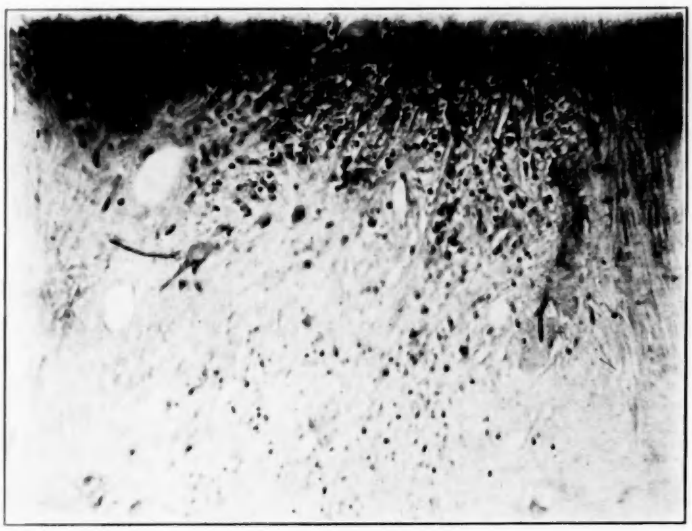

Fig. 2. Section stained with Verhoeff's modified gram stain showing organisms in tissue.

It would seem, however, that if this were true it would act upon each case alike. With the above patient there was no breaking down of the fibrous masses but a steady increase of fibrosis under intense potassium iodide therapy.

Cultural variations of the sporotrichum have been reported in the literature that may indicate various strains of the organism. It is noted that the French have reported some particularly malignant fatal cases of general sporotrichosis. It is possible that there is a type of the organism, most resistant to the usual medication. In this case a different form of therapeutic attack is indicated. The eosinophilia, involvement of the lymphatics and

indurated tumor masses of fibrosis in the above case indicate the usual tissue reaction to sporotrichosis. However, medication in this case, carried on for five years because of its reported value, had no effect.

Although X-ray, salvarsan and various local applications were tried it would seem that more experimental therapy should have been used.

Further laboratory study of the organism also would be of value.

Fig. 3. Section of culture media stained with Verhoeff's modified gram stain.

Radical surgery very early, with some method of sealing off the lymphatics as with surgical diathermy, would probably have offered the best results in this case.

Diagnosis.—Mistaken diagnosis may be more common in sporotrichosis than usually thought. In some parts of the country its rarity leads to forgetfulness, but it should be kept in mind. With only small subcutaneous nodules, particularly of the lids, the diagnosis of sporotrichosis may be confused with other lesions of similar appearance. If mistaken for the very common chalazion, it may be curetted without tissue examination and cause spread of a dangerous and possibly fatal disease. Every

small nodule of this kind should be studied with care and is best removed by complete dissection, if there is any doubt as to etiology. The best technic is required in its pathologic study. The first report on this case was "Giant Cell Sarcoma" (from a medical school pathologic department). With ulcerated lesions or discharging fistuli, cultural diagnosis is the method of choice.

CONCLUSIONS.

Due to the rarity of this disease it is essential that all available data should be considered carefully. The following points seem to present themselves for consideration.

1. While the literature is rather meager, sporotrichosis may be more common than realized and should be kept in mind.

2. The diagnosis is difficult with the nonulcerative nodules and may be confused with neoplasm or chalazia. Cultural diagnosis is the method of choice.

3. Too much confidence cannot be placed in potassium iodide. It has not always proved successful. Prognosis may be dubious.

4. Further study along the line of therapy is indicated. More radical surgery with some method of sealing the lymphatics, as with surgical diathermy, would seem advisable.

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DISCUSSION.

Dr. Arthur J. Bedell, Albany, N. Y.: Dr. Hill's case is one of unusual interest. It differs in two ways from the majority of the reported experiences: First, in the severity of the infection, and second, in its resistance to the iodine treatment. That does not mean that all cases have been cured with iodine. Dr. Hill has treated his patient for so long a time and with such large doses of the drug, both before and after operation, that we must conclude that the particular infection with which he is dealing is iodine resistant. His experience should cause us to approach a chalazion with more care, and all lid infections which fail to heal promptly should be subjected to a detailed bacteriological study. It is well to remember that the rare case in which sporothrix may be the activating agent can only be determined by accurate cultural methods, and further, that the diagnosis is more easily made by stained cultures than by the usual tissue examination. However, Verhoeff's new stain will help the pathologist to discover the sporothrix in the specimens submitted.

Dr. Hill has referred to our case, which was that of a 49-year-old man who had been suffering for two years with a constant discharge from his right eye. The skin of the right eyelids was congested. There was an irregular conjunctival mass 7x5x2 mm. on the nasal side of the upper right lid, extending 2 mm. beyond the ciliary margin. This was much like ordinary granulation tissue in appearance, structure and relation to the conjunctiva. The entire palpebral conjunctiva, especially the inner half of

both upper and lower lids, was congested with many discrete follicles and numerous 1 mm. shallow, yellowish-gray ulcers. The inner half of the bulbar conjunctiva was congested with several enlarged follicles. The caruncular fold was three times its normal thickness and uniformly infiltrated. Both puncta were prominent and dilated. The inner canthus was flooded with yellow, somewhat tenacious mucopus. After thorough irrigation, the discharge quickly reformed. There was no fullness of the lacrimal sac, and pressure over it caused no regurgitation from the puncta. The corneal epithelium was lost over an irregular 4 mm. area to the nasal side of the apex. The inferior and superior right ducts were opened and five firm, brown concretions removed from them and the lacrimal sac. The latter was thoroughly curetted. The sporothrix was found in culture after crushing the concretions. The conjunctival surface was painted daily with tincture of iodine and two grams of saturated solution of potassium iodide given three times a day. The recovery was uneventful. The small ulcers healed promptly, no new ones formed, and the lids were entirely smooth in ten days.

The sporothrix conjunctivitis sometimes closely resembles that of tuberculosis, lues or Parinaud's conjunctivitis. The cultural characteristics of the organism prove the diagnosis.

We found, just as did Dr. Hill, that the growth on secondary cultures was more abundant than on the primary. It must be understood that the organism is a strict aerobe.

The cases described by the Doctors Gifford are of prime importance, for they cover a group of some of the common ocular manifestations, especially the persistent lacrimal sac fistulæ.

We are indebted to Dr. Hill for describing the recent developments in his case. Ever since his first report we have wondered about the outcome, so that his paper today has proven unusually instructive.

ROSETTE FORMATION IN THE EYES OF IRRADIATED HUMAN EMBRYOS.*

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The rosette denotes primarily a structure of characteristic appearance occurring typically in retinoblastoma or neuroepithelioma (or neurocytoma) of the retina. The researches of Wintersteiner[1] virtually established the neuroepithelium of the retina as the basis for the new growth, and defined the rosette as a palisade arrangement of neuroepithelium about the external limiting membrane as a lumen. While some still believe that glial cells and their processes[2] rather than nervous cells furnish the material for the growth, Wintersteiner's conception of the rosette has remained almost universally accepted.

Rosettes in human eyes of other than pathologic significance have been found for the most part in embryos five to eight months old or in young infants. Seefelder[3] was the first to observe small rosettes in two fetuses, between the ganglion and inner nuclear layers; at the border of the optic nerve and retina in one case, and at the entrance of the nerve in the other. In the latter case the rosettes were derived from the nuclear layers and mostly surrounded by nerve and glial fibers. These were both otherwise normally developed eyes. A few years later Seefelder[4] found poorly formed rosettes in a pocket formation consisting of rudimentary retinal tissue in the edge of the disc in a newly born child. Mawas and Magitot[5] found a few rosettes in a six months fetus. Bertha Lindenfeld,[6] in 1913, described rosettes in the region of the nerve, together with folding in the outer retinal layers, in two fetuses six and seven months old, where X-ray radiation had been used in the second month of pregnancy to secure early abortion. These eyes were normal in size for this age, and were in all respects normally developed. Lindenfeld did not

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attribute pregliomatous significance to these changes. Likewise, Gilbert,[7] in describing rosettes in a case with congenital cataract and retinal anomalies, stressed that "glioma" does not occur in maldeveloped eyes. Pagenstecher,[8] in 1916, produced well formed rosettes in the retinæ of rabbit embryos which had been subjected to X-ray in utero. This experiment verified Lindenfeld's earlier observation regarding the formation of rosettes under the influence of X-ray.

Leber[9] held retinal rosettes to be due to pathologic processes in fetal life. One of Seefelder's cases had a fetal iritis, the other a high grade degeneration of the retina. Jaensch[10] pointed out

Fig. 1. Rosette in embryo of rabbit radiated experimentally. (From Pagenstecher, 40. Vers. d. Ophth. Ges., Heidelberg, 1916.)

that the rosettes found at the edges of colobomata and in folded retinæ of micropthalmic eyes (Wehrli[11]) are irregular in contour and occur in portions of the retina greatly altered. While the rosettes in his own case were well formed and regular, with a sharply demarcated limiting membrane, there were evidences of a previous iridocyclitis. Professor Fuchs[12] placed the developmental defect responsible for rosettes in the time when the greatest growth of the outer nuclear layer occurs, after three months. In his opinion, the infrequency with which rosettes are found in postnatal life merely indicates they undergo loss of their structure.

MATERIAL AND TECHNIC OF RADIATION.

The material to be reported consists of five cases, each the issue of a pregnancy in which, for one indication or another, large doses of X-ray were applied in the second month with a view to terminating the pregnancy. The first three ended at three and a half to four months; the fourth, in which radiation was applied over the spleen as a therapeutic measure in myelogenous leukemia, terminated at six and a half months (210 mm. embryo); the fifth ended unusually late, at seven and a half months (260 mm. embryo). In the last two cases, viable fetuses were obtained after artificial induction of labor, and each lived for one day. For their cooperation in facilitating this study we are indebted to Dr. Robert T. Frank of the Gynecological Service and to Dr.

Fig. 2. TECHNIQUE OF X-RAY RADIATION.

Case No.	Date of Exposure	Areas treated	No.	Skin Erythema Dose in %	K.V.	M.A.	Focal Dist. (cm.)	Time (min.)	Size of Field (cm.)
1	2½ mo.	Ant. abdomen	1	85%	200	4	40	29.3	22x22
		Post. field	2	85%	200	4	40	29.3	24x24
2	2 mo.	Ant. abdomen	1	85%	200	4	40	30.4	21x21
		Post. field	2	85%	200	4	40	30.4	23x23
3	2 mo.	Ant. abdomen	1	85%	200	4	45	30.5	22x23
		Post. field	2	85%	200	4	45	30.5	24x25
5	2½ mo.	Ant. abdomen	1	90%	200	4	50	48.6	20x23
		Post. field	2	90%	200	4	50	48.6	25x24
4		Radiation over spleen for myelogenous leukemia. 18 exposures of 20 and 25% skin erythema doses. Labor artificially induced at 5½-6 months.							
		Filter, ½ copper and ½ aluminum, all exposures.							

William Harris for his attention to the details of the X-ray radiation.

The anteroposterior diameter of the eyes of the last two embryos measured 11 and 13.5 mm., respectively, which corresponds with the normal dimensions for these ages. The three younger embryos having died in utero, some difficulty was found, owing to partial necrosis of the retina with dispersion of pigment through its layers. However, rosettes were seen in various stages of preservation. Those in Case 1 were particularly well formed. In Cases 4 and 5, the tissues were well preserved and allowed of easier study. In the former case, a single rosette was found close to the ora serrata and studied in serial section. Case 5 showed conversion of large portions of the retina into rosettes to an ex-

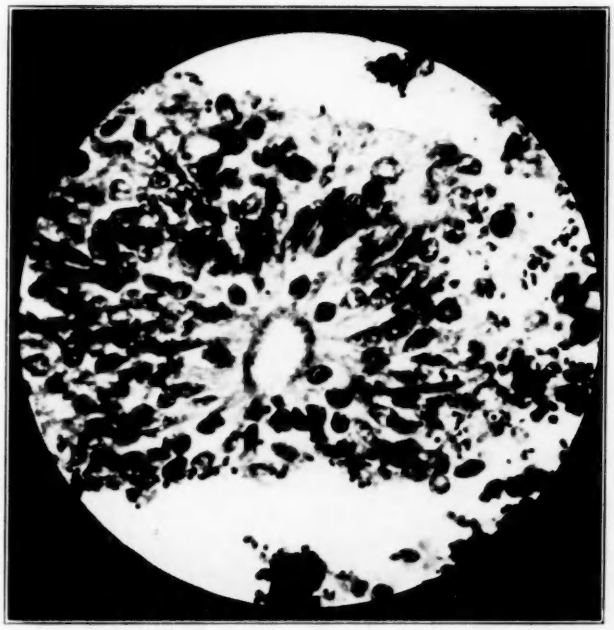

Fig. 3 (Case 1) (oil immersion). Rosette in outer retinal layers. Many processes project through the limiting membrane.

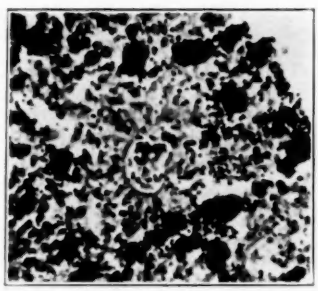

Fig. 4 (Case 2) (oil immersion). Poorly staining rosette, partially necrotic.

tent not previously observed in embryos. In addition, there were found failure of fusion of the upper portion of the fetal cleft, practically total conversion of the upper end of the nerve and disc into rosettes; and finally, faulty development of the ciliary processes and iris.

Fig. 5 (Case 3) (oil immersion. Portion of rosette. The limiting membrane is quite clear.

Fig. 6 (Case 4). The bodies of the developing cone cells, c, project through the limiting membrane; a few rods, r, are also seen.

MICROSCOPIC OCULAR STRUCTURE.

Case 1. A well preserved rosette is pictured in Fig. 3. It consists of a mass of round and oval nuclei about a clear lumen, bordered by a poorly defined membrane, through which project multiple fibrils. There are clearly defined cell bodies next to the lumen, roughly quadrilateral in shape, with deeply staining oval nuclei. Apparently these cells bear the processes projecting through the membrane. In position and appearance they correspond to the "cone cells" in their early stage, while the filament represents probably the beginning of the outer limb of the cone. (Mann[13]). The further developed cone cells with their processes in relation to a more definite external limiting membrane will be apparent in Case 4.

Case 2. An arrangement of cells about a membrane is shown in Fig. 4, in the midst of poorly staining retinal tissue.

Case 3. Here again (Fig. 5) a portion of rosette is shown, with a deeply staining limiting membrane. Theoretically, according to Mann, a definitive external limiting membrane is developed at this stage.

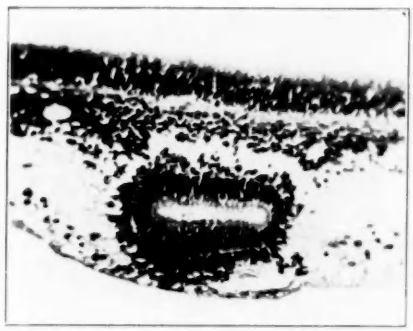

Fig. 7 (Case 4). Rosette in nerve fibre layer close to ora serrata.

Fig. 8 (Case 5). Non-union of fetal cleft at site of nerve. The cleft is lined with rosettes and poorly differentiated retinal tissue.

Case 4. A few cone and rod cells with their processes are depicted (Fig. 6) to show their elongated structure, the longer cell protrusions, and the relation of the cells to those of the outer nuclear layer. This structure is probably duplicated in the rosette seen near the ora serrata (Fig. 7). From within out are the lumen, the protruding outer limbs of the cone and rod cells, the external limiting membrane, the bodies of the cone cells with their oval nuclei, and the cells of the outer nuclear layer. Finally, there is a clear space, and a few cells of the inner nuclear layer grouped loosely about the rosette.

Case 5. Optic nerve. There is an apparent failure of fusion at one point in the upper part of the optic stalk, corresponding to the site of the disc. The cleft (Fig. 8) is lined on both sides with rosettes behind which is a cellular nerve fibre tissue. The cleft is not quite closed in Fig 9. In Fig. 10, at a lower level, there is complete formation of the disc and what purports to be the nerve, but which is in reality a fusion of elements similar to those lining the sides of the stalk seen in Figs. 8 and 9. Besides groups of rosettes, there are interlacing nerve fibres, ganglion cells and a few

Fig. 9 (Case 5). The cleft is not quite fused at a slightly lower level.

Fig. 10 (Case 5). Rosette structure of nerve (fused fetal cleft). C is a clear area resembling the plexiform retinal layer.

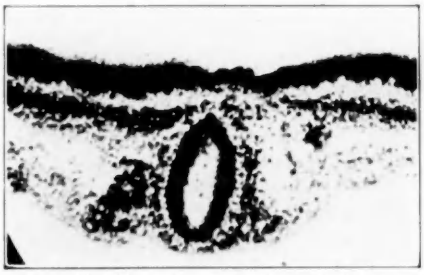

Fig. 11 (Case 5). A typical single retinal rosette.

Fig. 12 (Case 5). Multiple retinal rosettes; C, the clear areas, resemble the plexiform layer p, of the retina.

pigmented epithelial cells, togther with clear areas, such as c, in which the structure is fine and fibrillar, like that of the formed plexiform layer of the retina. There are no formed nerve bundles as in the fetal nerve of this age. A bit of tissue, bearing rosettes, has been detached and lies free in front of the pit.

Retina. Rosette formation is singly and in groups. Apparently only that portion of the retina close to the nerve, and at a level horizontal with it is affected. Rosettes are found in all retinal layers. Fig. 11 shows a rosette extending from the inner nuclear through the nerve fibre layer. The cells of the rosette resemble closely those of the outer nuclear layer, while about it are loosely arranged cells resembling those of the rarified inner nuclear layer. In Fig. 12 are multiple rosettes replacing the nuclear and nerve fibre layers. Where definite nerve fibres can be seen they appear to turn their course abruptly behind the rosette to assume the position formerly occupied by the outer nuclear layer (Figs. 13 and 14). This is not a folding of the retina but a genuine interchange in position of its layers. Fig. 15 shows foci of cells in the presence of intact or slightly disturbed nuclear layers, where rosettes are incompletely formed or poorly differentiated. In Fig. 16 are groups of rosettes separated by intervals of normal retinal tissue. At the ora serrata is a nodule (Fig. 17) projecting somewhat into the vitreous, composed of cells resembling those of the embryonal ganglion cell layer.

Uvea. The choroid is normal.

The ciliary body contains a fair number of muscle bundles in its flat portion, but there are no processes on its surface. Only posteriorly near the ora serrata are a few scantily developed pigmented folds. The iris is seen to spring from the margin of the optic cup, and is hardly more than a few layers of epithelium. (Figs. 18 and 19.) The marginal sinus M is

Fig. 13 (Case 5). The deviation of the nerve fibre layer, f, posteriorly, behind the rosettes.

Fig. 14 (Case 5). Total absence of layer structure, with rosette structure in front and nerve fibres behind, next to choroid, ch.

the space between the two layers of the optic cup. Behind Descemet's is a loose vascular tissue which in one or two places covers the edges of the rudimentary iris, but no iris stroma as such is made out. An anterior chamber can hardly be said to exist.

Lens. The lens has been displaced into the vitreous. Those sections containing it show it to be of normal size, contour and development, and there is no evidence of cataractous or other change.

COMMENT.

The eyes of five embryos are presented, in each of which rosette formation occurred under the influence of X-ray radiation in rather large doses. From this study and from the evidence contributed by Lindenfeld and Pagenstecher, it may be adduced that the development of rosettes is a specific result of this type of radiation on growing retinal tissue.

The rosette pictured in Fig. 3, in a three and a half to four months embryo, represents the earliest stage in fetal life in which this structure has been described. While the specimen does not allow of distinction between the inner and outer neuroblastic layers, a rather systematic arrangement about a limiting membrane is evident, consisting of cells with oval nuclei. These are

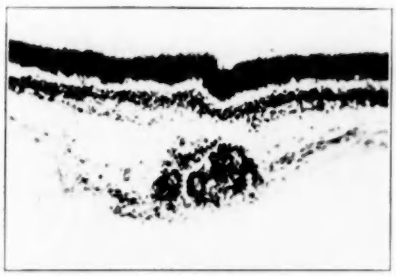

Fig. 15 (Case 5). Poorly differentiated rosettes in anterior retinal layers.

Fig. 16 (Case 5). Foci of rosettes occurring at intervals in the retina.

very probably the outermost cells of the outer neuroblastic layer, while the filaments piercing the lining membrane correspond to the cone processes which represent the inner limb of the definitive cone. According to Mann, the external limiting membrane is present at this stage. The rosette in Case 4 represents merely an advanced stage of cellular growth and differentiation involving the cone and rod cells, the cone filaments and the outer neuroblastic layer.

In addition to formation of rosettes in Case 5, there are a number of anomalous conditions which form interesting material from a developmental point of view. Granted that the rosettes are attributable to radiation, one must still account for the persistence of the fetal cleft and for the rudimentary state of the iris and ciliary processes. One cannot entirely exclude the possibility of an inherently defective eye in which a severe stimulus such as X-ray has caused secondary changes as rosette formation. Lindenfeld emphasized that both of her cases showed no anomaly of development other than rosette formation. Still, one must seriously consider the anomalous conditions in this case, as well as the rosettes, as being due to the application of radiation at a

Fig. 17 (Case 5). Nodule in retina at ora serrata.

critical period in development, namely, during the second month of fetal life. Moreover, in considering the unparalleled extent to which retina and nerve are involved in the change to rosettes, one must bear in mind the massive dosage of the present day, as compared to the smaller doses which were prevalent at the time Lindenfeld reported her cases (1913).

The persistence of the cleft at one edge of the disc, lined with tissue bearing well formed rosettes, together with the almost total conversion of the nerve into groups of rosettes (Fig. 10) forms a picture that is without precedence in the literature. The rosettes described in and adjoining colobomatous areas are hardly as well formed or as numerous. Those in microphthalmic eyes, though often well formed, are likewise few in number, and accompany folding in the outer retinal layers. The dimensions of the eye in this case are normal for this stage, although the possibility of microphthalmus, had the embryo grown to full term, cannot be excluded.

The anomalous conditions aside from the rosettes enumerated in the description—persistence of the cleft and rudimentary development of ciliary processes and iris—speak in general for primary and secondary effects of the radiation. The initial effect may be considered as occurring at the time of radiation, in this case during the second month. According to Mann, the fetal fissure has closed before the 20 mm. stage (seven weeks). It can be conceived that the application of an intense stimulus at this period inhibited complete closure. A similar influence apparently

Fig. 18 (Case 5). Anterior segment, with rudimentary iris, I, and slightly developed ciliary processes, P. The lens has been displaced in fixation.

worked upon the rest of the inner wall of the optic cup to the extent that the ciliary processes, which make their appearance in the third month, have retained a primitive state at seven and a half months. Likewise the iris is no further advanced than the four and a half months stage, having just budded off the edge of the optic cup. This cessation of further differentiation of portions of the inner wall of the optic cup could be considered a secondary effect of the radiation, owing to the latency and prolonged effect of X-rays in tissues.

Professor Fuchs, in discussing Pagenstecher's[14] experimental work with rabbits, spoke of the excessive growth of the primitive outer neuroblastic layer as being responsible for rosettes. Undoubtedly this layer, which furnishes the nuclei of the cones and rods, and finally differentiates into the outer nuclear layer, is responsible for the bulk of these structures. However, the multiplicity of rosettes throughout all retinal layers, in the absence of a definitive outer neuroblastic layer (Fig. 12) suggests a destructive, in addition to an alterative influence of the radiation. Here is only a mass of nuclei in place of the regularly formed layer. Most interesting is the turning of the nerve fiber layer behind the rosettes (Fig. 14). Its significance can only be speculated on.

It will be seen, therefore, that the ectoderm, and more specifically the inner wall of the cup, is involved in the changes enumerated. The choroid, the muscle of the ciliary body and the pupillary membrane (mesodermal portion of the iris) show no abnormalities. The eye is of normal size. Apparently, then, the mesodermal elements are intact.

The case under consideration presents more than theoretical interest as regards a possible relationship to retinoblastoma.

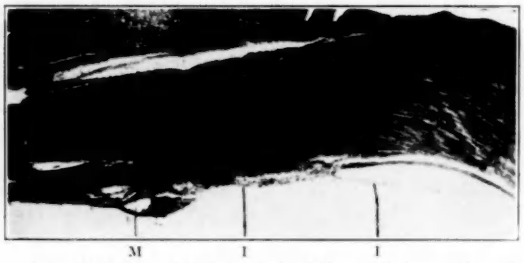

Fig. 19 (Case 5). Iris, I, budding off from the edge of the optic cup. M is the marginal sinus.

Deep X-ray therapy has been used with some success in the treatment of retinoblastoma. We have then the strange coincidence of the same physical agent first evoking rosettes and then being responsible for the destruction of this structure which contributes an important if not basic feature of the new growth—in the first instance a stimulating, in the second, a destructive influence. The presence of anomalies in this case detracts from the possibility of tumor formation in the event the embryo had survived, since it is virtually established that retinoblastoma does not develop in anomalous eyes.

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DISCUSSION.

DR. E. V. L. BROWN, Chicago: I have very little to add to this excellent presentation of the subject.

The author has undoubtedly demonstrated the formation of rosettes at a very much earlier stage than had ever before been seen in microphthalmic eyes or glioma. E. Fuchs believes that these rosettes are the result of overstimulation of the retinal layers. The author speaks of the inhibition of closure by the mesoderm, as held for decades as the genesis of the condition. In this connection I wish to call attention to the recent lectures of Dr. Ida Mann, in this country, and to present a chart explaining a more recent view, namely, that the condition is due to overstimulation, which would be in exact keeping with the author's findings.

The first drawing shows retinal layers in normal closure. According to the author's view, closure would be prevented by persistence of the mesoderm at this point. Dr. Mann has not found this to be the case, but that the overgrowth of the retina downward meets and blocks the ends of the pigment epithelial layer; thus the overgrowth of the inner layers of the retina occlude the pigment epithelium at a point farther back from the center of the cleft than are the layers of the nuclear portion of the retina proper.

DR. WILLIAM C. FINNOFF, Denver: There is one point in which I wish to differ from Dr. Goldstein, and that is the statement he made regarding rosettes in glioma or retinoblastoma as compared with the rosettes shown here today. I think the rosettes he has shown are identical with the rosettes seen in retinoblastoma. The true rosettes in retinoblastoma represent the outer ectodermal layers, the layers of rods and cones with the external limiting membrane, and the statement that in retinoblastoma, or so-called glioma, a blood vessel is found in the center of the rosette is inaccurate. The center of the rosette in retinoblastoma is exactly the same as that in the rosettes just shown—i. e., a group of protoplasmic processes radiating from an avascular center with the nuclei toward the periphery. There is an erroneous impression held, I think, by many pathologists, that the central blood vessel surrounded by cells is a part of the rosette formation. These rosettes were first observed in so-called glioma by Flexner and not by Wintersteiner as is commonly believed.

It will be very interesting to radiate embryos at the time of the very earliest stage of differentiation of the ectodermal layers into the retina to determine whether rosettes form from the outer layers of the retina just at the time that they appear. The differentiation of the rods and cones occurs late and takes place first in the posterior portion of the globe, then progresses forward. This late formation of the layer of rods and cones in the embryo might explain the absence of rosettes in the region of the ora serrata, and the very frequent formation of these structures in the posterior portion of the globe in Dr. Goldstein's specimen.

I think that this has been a very valuable work and probably will throw some light on the etiology or the method of development of these malignant tumors of the retina.

A STUDY OF CALCIUM IN RELATION TO CATARACT: REPORT OF RESEARCH FELLOW IN OPHTHALMOLOGY.

DANIEL B. KIRBY, M. D.,

NEW YORK.

Many empirical studies have been made with the hope of discovering something which would prevent the dreaded onset and progress of cataract or restore useful vision to those afflicted. It is a fact that in certain eyes there are observed opacities which have developed to a certain stage and then because the factor which produced the opacity has become inactive the cataract no longer progresses. The opacity is pushed toward the center of the lens as more clear cortex is formed by the equatorial cells. The observation of this phenomenon time and again, rather than the reports of spontaneous absorption or retardation, arrest or cure of cataracts by various local and systemic measures constitutes a valid reason for continuing the study of the nonsurgical treatment of cataract, both from the clinical standpoint and by fundamental investigations into the nutrition and metabolism of the crystalline lens.

It has been recognized that the manifestations of cataract in its various forms are quite different and undoubtedly dependent on various causes. Congenital and infantile forms are really in a class by themselves. Senile cortical cataract is quite apart from the senile nuclear type, even though it may be superimposed upon the latter, and again they both differ from the posterior cortical form. Separate, also, is that rapidly progressive variety, where the changes occur directly beneath the anterior and posterior capsules and end with diffuse clouding of the cortex and nucleus.

Acknowledgment is gratefully made to the American Academy of Ophthalmology and Oto-Laryngology for the funds which were supplied the Research Fellow in Ophthalmology. Professor John M. Wheeler procured an additional necessary grant from an anonymous donor. The work was conducted in the Department of Ophthalmology at the College of Physicians and Surgeons, Columbia University, in the Vanderbilt Clinic and Presbyterian Hospital.

Acknowledgment is made to Newell W. Giles, M. D., for his cooperation and assistance in the clinical investigation.

ANATOMY OF PARATHYROID GLANDS.

The parathyroid glands in the human are bean-shaped bodies, 3 to 15 mm. long and 2 to 3 mm. broad and thick. They are usually four in number. They are developed from the third and fourth pharyngeal pouches and are situated within or on the posterior surface of the thyroid gland. They are composed of columns or masses of epithelial cells.

EXTRACTS OF THE PARATHYROID GLANDS.

Various extracts of the parathyroids have been prepared and more or less success reported through their use in combating signs and symptoms of tetany. Collip[5] finally brought forward proof that by weak acid hydrolysis of the parathyroids of the ox there could be obtained an active principle, which was called parathormone, and which was found to uniformly and consistently prevent or relieve tetany in thyro-parathyroidectomized dogs. The probable manner of action was through a direct effect on the calcium metabolism. The injection into normal animals causes a definite rise in blood calcium, and it is by this means that the product is assayed. Evidently the physiologic action of the parathyroid glands is to maintain a definite level of blood calcium in the circulating blood. The estimated normal daily production of the hormone by the human body is about 45 units. A unit of potency has been defined as one one-hundredth of that amount of extract which produces on the average a 5 mgm. per 100 cc. rise in blood serum calcium of 20 kilo-dogs, after 15 hours. (Collip).[5]

A Study of Calcium Deficiency and Its Relation to Cataract.

TETANY.

Zonular cataract is found, above all, in children who have suffered from convulsions. (Arlt).[11] At the same time there are found peculiar changes in the teeth (Horner),[11] which, together with the cataract itself, are regarded as due to tetany (Peters and others),[11] although formerly both were attributed to rickets. The lens, which in its development has much in common with the teeth, is also supposed to undergo some disorder during a period of its development, while later, after the disappearance of the disorder, normal transparent layers of lens substance are again deposited. Hess[12] quotes Dick, who in cases of zonular cataract

found a very large percentage with hypoplasia of the dental enamel. In tetany, as result of parathyroidectomy, calcium may be has been recognized. Much has been learned concerning the complex relationship of calcium to other elements and salts and of its importance in the maintenance of colloidal solutions, such as lens protein.

SOURCE OF CALCIUM.

Calcium is taken into the body in the form of organic and inorganic combination from milk, cheese, butter, eggs, green vegetables and nuts. Digestive changes convert it entirely into the inorganic form, so it is absorbed from the intestine as partly or completely ionized. An excess in the diet is desirable. The adult requires 0.4 to 1.0 gr. daily. Until the growth of the skeleton is complete, the daily requirement is greater.

The ash constituents of the dietary are certainly of importance. In fasting there is a constant loss of salts from the body. "Wear and tear" metabolism of the bones must be replaced by ingested salts. The ordinary American diet of the average inhabitant of the Eastern States, McCollum[6] states, presents a sorry spectacle in regard to calcium content. Sherman[6] and his school have continued unceasingly to advocate a proper intake of calcium in the dietary. They report that the calcium of vegetables is not so efficiently utilized as that of milk, and they commend a quart a day as advisable for each member of the family.

EXCRETION OF CALCIUM.

Calcium is eliminated from the body chiefly by the large intestine and to a lesser extent by the kidney. Urinary excretion varies from 0.1 per day on low calcium diet to 0.5 gr. on a normal diet. Acid forming diets increase the amount excreted by the kidney.

CONCENTRATIONS OF CALCIUM IN BODY.

The calcium of the blood is almost entirely in the serum. The level of the blood serum is normally about 9 to 11 mgms. per 100 cc. It is there combined with protein and various anions. About 54 per cent of it is diffusible—that is, it is in inorganic form and can pass through an animal membrane. This is the form which is found in the spinal fluid and in the aqueous and vitreous humors. Collip[5] feels that it is impossible to say how much of the calcium in the blood is ionized and how much is nonionized, but more re-

cently Hunter[3] has found the proportions to be: (a) ionized, diffusible, 2 mgms.; (b) nonionized diffusible inorganic, 3.5 mgms., an (c) nonionized, nondiffusible organic, 4.5 mgms. per 100 cc.

CALCIUM IN AQUEOUS AND VITREOUS.

Duke-Elder[8] has found the average amount of calcium in the aqueous to be 6.2 mgms. per 100 cc. and that of the vitreous 6.8 mgms. per 100 cc. of material obtained from horses' eyes. Cohen found an average of 7.4 mgms. calcium in the aqueous and 7.9 mgms. in the vitreous of oxen eyes. The extremely small amount of human aqueous and the element of risk attached to securing it has rendered data from this source meager and unreliable. It may be assumed, however, that the ratio holds true in the human eye and that the aqueous and vitreous contain a little more than half (5 to 7 mgms. per 100 cc.) of the amount of calcium found in the blood serum (9 to 11 mgms. per 100 cc.). This is comparable to the amount contained in the spinal fluid. In many ways, in mode of formation and in composition, the aqueous resembles the spinal fluid.

CALCIUM IN THE LENS.

Burge[1] found the calcium content of the normal lens too small to estimate. In the cataractous lens (U. S. A.) there was 12.5 per cent of calcium in the ash, while in the Indian cataracts there was 6 per cent. Adams[2] figured the former was equivalent to 0.21 per cent of the dry weight, while the latter was 0.09 per cent of the dry weight.

Adams[2] found an average of 0.0516 gms. calcium per 100 gms. dry weight of the crystalline lens, while that of the human cataractous lenses was 0.1265 gm. calcium per 100 g. dry weight. She found no definite age-variation in the calcium content of normal or cataractous lenses. She confirmed Burge's[1] results but found a considerable variation in the calcium content of individual cataractous lenses, the deposition of calcium in cataract being therefore an erratic process. Frozen sections of cataractous lenses showed irregular deposition of calcium in the individual lenses. One in every ten or fifteen showed fairly large circular deposits of radiating crystals. These have been described by Wessely and Kranz,[2] who found them to be crystals of calcium phosphate and who thought that they represented a local deposit of calcium in areas where a preceding colloid or protein degeneration had oc-

curred. Adams used a number of staining reactions and confirmed the presence of calcium.

CALCIFICATION OF THE LENS.

Calcification of the lens may occur within the intact capsule and is often found in the final stages of complicated cataract, in shrunken globes, and sometimes in diabetic cataract. Partial calcification is also seen in old anterior capsular cataract.

THE FUNCTIONS OF CALCIUM.

Calcium enters into many physiologic processes. *Bone:* The most evident function of calcium in the body is the formation of bone. *Coagulation of Blood:* Calcium in ionized form is necessary for the coagulation of blood. *Nerve and Muscle Tone:* Calcium with other ions has to do with the maintenance of a balanced system which determines the irritability of nerve and muscle. Calcium has a depressor action, lessening the irritability. *Epithelium:* Calcium is concerned in the healthy state of epithelial cells and their products. *Endosmosis:* Calcium has a tendency to diminish the permeability of animal membranes and in this connection undoubtedly has much to do with the endosmosis or passage of fluid through the semipermeable membranes of the ciliary epithelium and the lens capsule. *Colloids:* On salt concentration depends the maintenance and stability of colloidal systems. This is interesting when one considers the rôle of calcium in that unique colloid, the transparent lens protein. *Acid-Base Metabolism:* Aub has shown that calcium is the most available body base and that it is easily drawn upon in time of need to counteract acid ions and maintain the acid-alkali balance of the body. The calcium ion is very important in this respect, as sodium and potassium are protected. The storehouse for calcium is in the meshwork of spicules of the bony trabeculæ which ramify through the bone marrow. Studies by Gamble[7] showed that during fasting calcium is the only fixed base which is drawn from the body tissues. *Respiration and Fermentation:* In the lens the acid-alkali balance has, indirectly, much to do with the normal respiration as also with the autoxidation system and the process of hydrolysis of ferments as described by Burdon-Cooper.[10]

There are other normal functions of calcium in the living body. The functions under abnormal conditions will be considered in another section.

THE CONTROL OF CALCIUM METABOLISM IN THE BODY.

The absorption, storage, mobilization, blood and tissue level and excretion of calcium is not accomplished in a haphazard way. It is in part controlled by the physiologic needs of the tissues, but the remarkably steady level of the calcium in the circulating blood is maintained by the action of the secretion of the parathyroid glands.

One avenue of approach to the subject has been through the study of the inorganic salt metabolism, as normal growth and senescence are fundamentally dependent upon the normal or abnormal function of this mechanism. Inorganic elements play an important rôle in the makeup of the crystalline lens protein.

There has been known for some time the association of cataract with infantile and other forms of tetany and parathyroid deficiency, conditions in which it was definitely recognized that the calcium metabolism was disturbed. Several investigators have also considered senile cataract to be associated with latent tetany and parathyroid deficiency or with senile decrease in function of other endocrine glands.

Fischer and Triebenstein[1] examined 68 cases of senile and presenile cataract and claimed to have found evidences of tetany and latent tetany in 88.2 per cent. Tron, A.,[2] and S. V. Pellathy[2] and Polichova,[2] using different methods, studied the blood serum calcium in senile cataract patients but could not confirm the association with parathyroid insufficiency. There was, however, considerable difference in their results and in their comparison of cataract cases with their normal.

O'Brien[3] found serum calcium values within normal limits.

Adams[2] compared 20 cases of both sexes in normal subjects with 20 of those who showed senile cataract and found 9.76 mgms. of calcium per 100 cc. blood serum for the controls and 11.46 mgms. per 100 cc. for those with senile cataract. This was evidently an increase of calcium in the serum of the latter rather than a deficiency.

Burge[4] found that the calcium in the crystalline lens increased from a very small amount in the normal to a relatively large amount in cataract, 12.5 per cent of the ash of the cataract. Adams found the calcium content of the normal lens 0.0516 gms. per cent dry weight for the normal and 0.1256 gms. per cent dry weight for the cataractous lens.

So much has been learned concerning calcium metabolism recently that it seems worth while to revive interest in the subject and pursue a complete study in regard to the relation of calcium to the eye, and particularly to the lens and cataract. It has been proved that the secretion of the parathyroid controls the calcium metabolism. Collip has discovered and isolated for clinical use the active hormone of the parathyroids. Viosterol and Vitamin D and their action have been discovered. The effect of sunlight and ultraviolet light and other activators on calcium metabolism as low as 2 to 3 mgms. per 100 cc. of blood serum, while in latent tetany, demonstrable only by altered electrical reactions and by the Chvostek sign, the calcium content varies between 6 and 10 mgms. When below 5, convulsions occur. In rickets, however, in absence of tetany, the blood calcium is seldom lowered. There have been observed marked cases of rickets with normal calcium content. Cases of rickets that have cataract have also undoubtedly had tetany.

RICKETS AND TETANY.

It will be interesting in this respect to compare rickets and tetany, conditions which are often associated in the same individual, but which are not identical or dependent upon the same factors. The history of spasms or convulsions, especially as stressed in the etiologic relationship to zonular cataract, would point to its belonging to the symptom complex of tetany rather than to rickets.

Hess[12] has found that although tetany may occur occasionally in an infant free from rickets, from a clinical standpoint all infants with signs of tetany have rickets to some degree. But the contrary does not hold true, for many cases of rickets, even some of the most severe cases, show no evidences of tetany. Tetany has long been regarded as a complication of rickets. In infants the same factors of diet and environment contribute both to rickets and to tetany and the same treatment relieves both conditions.

As Hess[12] has pointed out, it is of little value to theorize about the relationship of, or the differences between rickets and tetany. The question is being actively studied in the clinic and laboratory now and further information will be forthcoming. At the present time, as far as can be observed, the occurrence of cataract is linked up with the tetany complex rather than with that of rickets.

Zonular cataract is observed usually in children of the early school age, but its origin probably goes back to the early infancy of the child.

TYPES OF TETANY.[3]

Tetania parathyroipriva—deficient function of parathyroid.

Tetania strumipriva—postoperative removal or damage, or cutting off blood supply of the parathyroids.

Tetany of alkalosis—blood calcium normal.

Tetany of hyperpnea.

Tetany of pregnancy.

Tetany of idiopathic or unknown origin.

LESIONS OF ECTODERMAL TISSUES.

Cataract, brittleness and ridging of the nails, loss of hair and defects in the enamel of the teeth have been described in tetany.[13] Cataract is seen four times as frequently as the other three changes.

The association of cataract with tetany was described about 50 years ago. It has been observed in endemic tetany, postoperative tetany, tetany complicating rickets and spontaneous hypoparathyroidism. Thyroparathyroidectomy in dogs may produce opacities in the lens within periods varying from three months to two years. Peters[1] thinks that cataract, following tetany, is due to a shrinking of the nucleus brought about by interference with the normal nutrition. His suggestion is that cramp of the ciliary muscle, which occurs in the course of the fits, causes an irregularity in the flow of the lymph.

INFANTILE TETANY.

Tetany occurs generally in bottle-fed, very rarely in breast-fed babies. There is usually a gastrointestinal disturbance with constipation. It is more serious in a premature infant than it is in a child of four months to two years of age. Pediatricians are trained to look for the slightest signs of latent tetany, and all infants are regularly placed on antirachitic and antitetany measures so that the cases are much fewer than formerly and those that do occur are better controlled.

At the Babies' Hospital, New York, 33 cases of tetany were treated during the past two years. The records of 27 of these patients were available to the author. Eighteen were male, nine female. They ranged in age from two weeks to eighteen months.

Twenty of these cases had antecedent rickets. The blood calcium ranged from 4.2 to 8.0 with an average of 6.1 mgms. per 100 cc. of blood. Except for two of the infants, who died from intercurrent disease, the patients were uniformly improved by the measures taken to improve the calcium metabolism. In no case was cataract present or developed during the period of observation. There were no readmissions because of recurrence of the tetany. Undoubtedly these conditions now prevail throughout the country. The better understanding of the nature of tetany, its prevention and cure has led to a great reduction in the number of tetany cataracts.

INCOMPLETE TETANY CATARACTS AS SEEN IN LATER YEARS.

Quite often zonular or lamellar cataracts are seen where it is evident that the factor which caused the opacity has ceased to act. Clear cortex has been formed and has pushed the opacity toward the center of the lens. Two such cases were observed, one that of a boy of nine years and the other a man of thirty-seven. In both of these, the serum calcium was normal and there was no evidence of latent tetany.

POSTOPERATIVE TETANY WITH CATARACT.

Reports of 38 cases of postoperative tetany with cataract have been published to date.[13] A review of the two more recently reported cases will be interesting. *Hunter's case*:[7] a case of postthyroidectomy in which tetany developed, in a single woman of 44 years, was cited by Hunter. The serum calcium fell to 6.8 mgms. per 100 cc. of blood serum. The lenses, as examined by Goulden, showed no opacities with the ophthalmoscope, but with the microscope three types of opacities were seen. One was flaky white and situated just beneath a clear area of cortex which separated it from the capsule. Another type was angular and iridescent, usually blue or red, and in the same location; the third was a few powdery opacities in the depth of the lens in the fetal nucleus. The cataracts did not progress to complete opacity. *Cole's case*:[13] The case was that of a woman of 34 years who had developed goiter at the age of 18 years. One year later, some glands were removed from the side of her neck. At 33 years, the goiter had grown so large that it had to be removed. In February, 1928, partial thyroidectomy was performed, the middle lobe being left. A week later there was stiffness of fingers and difficulty

in the use of several other muscles. Her hands would become cramped. In July, 1928, she had a fit, and lost consciousness for one hour. She then first complained of dim vision, lasting for one hour. Two other fits were experienced in the same month, and each was followed by dimness of vision. No more fits occurred for five months, then in December, 1928, she had them frequently, several a day, continuing until March, 1929, when they stopped. The misty vision did not pass off quickly after the fits in December, and it gradually became continuous. On March 23, 1929, Chvostek's sign was very pronounced. Trousseau's sign could not be elicited. Her vision was greatly diminished. The serum calcium was 3.2 mgms. per 100 cc. blood. She was given milk and eggs and calcium lactate. Her eyesight grew steadily worse, and on April 4, 1929, she was almost completely blind.

The patient appeared robust and healthy. Mucous membranes, hair, nails and teeth were normal. The eyes showed fairly generalized cataracts, both cortical and nuclear, laid down in large pyramidal masses, with bases toward the periphery. These were of no great density, but sufficient to obscure the red reflex without obliterating it entirely. From Cole's description and from the illustration, one can discern a diffuse clouding with split sutures forming the pyramidal masses described. X-rays showed nothing abnormal in bones or teeth. High calcium diet and calcium lactate had a marked effect in raising the serum calcium and controlling tetany, but did not bring it to the normal level until combined with parathormone. In comparison, dried parathyroid extract was given in doses as advised for ten days with the same calcium intake and the serum calcium fell to 4 mgms. and there was a return of the signs of tetany.

COMMENTS ON COLE'S CASE.

It is worth while noting that Cole described a very marked increase in the lens opacity following an increased calcium diet without parathormone. In the first two weeks of treatment the cataracts went on to complete opacity in both eyes while the tetany disappeared. This would support Greenwald's theory that in the absence of parathormone hormone, there is an extensive deposit of calcium in the tissue, probably in the form of calcium phosphate. As a precaution, as Cole has advised, it may be wise in severe parathyroid tetany to give parathormone before administering calcium intravenously or before increasing the amount of

calcium by mouth. Then the parathormone can control the deposition and excretion of the added calcium. It may be added further, however, that parathormone had no effect in retarding or clearing up the cataract in this case.

CALCIUM THERAPY A VALUABLE ADJUNCT TO HORMONE IN CASES OF DEFICIENCY.

Adams[2] found that by giving calcium lactate orally to a rabbit it was possible to raise the blood calcium. She gave 1 gm. of calcium per day to normal animals and continued for five to six weeks, and found no evidence of cataract, although within two hours of each dose the serum calcium rose 6 to 7 mgms., and after eight hours was still 2 mgms. above normal, while it regained the normal level in 20 to 24 hours. She also proved that oral administration causes a corresponding rise in the aqueous humor. In other animals, she found that the administration of calcium did not in any way accelerate the development of cataract which is readily caused by naphthalene.

Aub[14] has found that humans showing only slight response to the parathormone have shown marked increases in the serum calcium to as high as 19 mgms. per 100 cc. when calcium lactate was added to the diet. It is not entirely logical to feel that the use of increased calcium intake before parathormone in Cole's case was the cause of the rapid progress of the cataracts. This may have been entirely a coincidence. Before Collip isolated parathormone the parathyroid extracts were more or less ineffective, so that a number of cases of postoperative and infantile tetany have received calcium in increased amounts by mouth without having cataracts develop or progress. However, in view of Cole's experience, it may be well to give parathormone first.

PROGNOSIS FOR VISION IN TETANY CATARACT.

The prognosis for vision in such cases depends upon the removal of the cataracts. There is no reason why this should not be successful. In Cole's case the operation had not been completed.

CATARACT COMPLICATING POSTTHYROIDECTOMY TETANY REMOVED SUCCESSFULLY.

Reeder[15] published what appears to be still another case of cataract following thyroidectomy. It was evidently caused by the coincident removal of the parathyroids. The patient was a fe-

male of forty years, who six months from the time she developed goiter with toxic symptoms had her thyroid removed. A few days after the operation she developed tetanic spasms which were arrested by large doses of calcium lactate and parathyroid. These were given at intervals for over a year. Her tetany practically disappeared six months after the thyroidectomy. Eighteen months later she had a well advanced cortical cataract in each eye. Removal of the cataract from each eye gave good visual results.

THE PATHOLOGY OF TETANY CATARACT.

The work of Siegrist[16] and Goldmann has shown that the opacity in parathyroidectomized dogs is always at first subcapsular, and appears in close relationship with tetany attacks.

The capsular epithelium shows degeneration. The form of the cell is changed, the cells appearing necrotic. The nucleus changes in form and staining. However, here and there a mitotic figure is observed. Disintegration is visible in the superficial lens fibers, empty spaces appearing between them which are filled with either minute fat or protein droplets or homogeneous substance.

Such changes are not found in the lens nucleus. The ciliary epithelium shows no pathologic changes.

PATHOGENESIS OF TETANY CATARACT.

Verhoeff[17] placed the lens removed from a 3½ weeks old human infant's eye, which had been fixed in formalin, in 1 per cent osmic acid for five days. The lens was first bisected to ensure penetration of the solution. On microscopic examination there was found a peripheral zone, thickly studded with blackened droplets. These droplets were readily bleached when treated with a saturated solution of potassium permanganate followed by oxalic acid, thus proving that they were not ordinary fat but that they were probably related to myelin. If these findings are confirmed by further observations, they will prove conclusively that lamellar cataract is due to the undue persistence of lipoid droplets normally present in fetal lenses. Their persistence in cases of congenital cataract possibly could be explained by a faulty fat metabolism, either dependent on or associated with the faulty calcium metabolism. Ultimately it is possible that they may be replaced by lime salts or other substances.

To explain the probable relation of coralliform cataract to faulty calcium metabolism seemed to Verhoeff difficult. Professor Osterhout informed Verhoeff that the conditions under which the pro-

teins crystallize in living tissues are not definitely known but that almost any protein may be made to crystallize out by withdrawing the salts from the solution in which it is dissolved. The possibility suggests itself, therefore, that the crystallization of the lens protein in corralliform cataract may be dependent upon a deficiency of calcium or other salts in the blood or aqueous humor.

It is difficult to prove the mode of action of decreased calcium content of serum, and necessarily of the aqueous, vitreous and lens in the pathogenesis of tetany cataract. In a case of an infant with rickets or in a preoperative thyroid case, the serum calcium level is normal. Then in the infant some unknown factor spontaneously reduces the serum calcium, while in the operative case the coincident removal of parathyroid tissue with thyroid or the circulatory disturbance of the former with associated temporary or permanent diminution in function of the parathyroids likewise reduces the serum calcium and causes tetany. What has become of the calcium represented by a drop from 10 mgms. to 5 mgms. per 100 cc. of serum? In a person of 60 kilos. body weight, there are approximately 5,280 cc. of blood. A drop of 5 mgms. per 100 cc. would be equivalent to the loss of approximately 264 mgms. of calcium from the blood. Is this deposited in the tissues, as, for instance, in the lens, or is it excreted? To answer this, one must consider the action of the secretion of the parathyroids which controls the serum calcium level. After ingestion of calcium salts or calcium from the diet, the absorbed calcium goes into storage or is excreted without appreciably altering the serum calcium level in the normal person. There is a relatively tremendous turnover of the calcium each day, there being absorbed and excreted more than the total amount present in the entire blood of the individual at any time. The drop in serum calcium level of parathyroid deficiency then means that while excretion goes on there is a decrease in the absorption or in the mobilization of calcium from the normal storehouses. The amount represented by the decrease simply has been excreted and is not replaced.

The lowered serum calcium level means that there is a proportional diminution of the calcium of the aqueous, vitreous and lens. While this total amount is extremely small, the electrochemical changes and the disturbance of equilibrium, depending on the loss of these calcium ions is out of all proportion to the chemicals involved. It can certainly cause changes in the labile lens protein, hydrolysis with some evidence of hydration in the

form of globules, vacuoles, cloudy swelling of the lens fibers occurring as a result in the change in hydrogen ion concentration.

PREVENTION OF POSTOPERATIVE TETANY AND CATARACT.

The prevention of removal of the parathyroids with thyroid tissue is important. It is best accomplished by leaving the posterior and mesial portions of the lateral lobes. This precaution will usually preserve even the glands which are placed in anomalous positions. In a series of about 500 cases at Presbyterian Hospital, Parsons[18] has had only three cases of considerable parathyroid deficiency and three cases of mild disturbance. All thyroidectomy cases are watched carefully after operation, and if they show the first premonitory symptom or circum-oral constriction or difficulty with speech or with moving their lips, this symptom coming on before the Chvostek sign is positive and before they have any carpopedal spasm they are given calcium lactate (up to 30 grains daily) and parathormone (15 units daily). Only one case failed to respond fully to this treatment, and even she has not developed any severe tetany or any cataract. In the Lahey clinic[13] the thyroid tissue is carefully examined at the time of operation, and if the parathyroids have been removed they are grafted into the sterno-cleido-mastoid muscle. This technic has produced a living graft in animals.

FACTORS USED IN JUDGING THE CALCIUM METABOLISM OF CATARACT PATIENTS.

In order to judge the calcium metabolism of patients with cataract, from all possible angles, all factors bearing on this function were investigated.

COAGULATION AND BLEEDING TIME OF BLOOD.

Ionized calcium[19] is necessary for the coagulation of the blood. Only a small amount is necessary for normal coagulation, and rarely, if ever, does the calcium in the serum fall to such a level as to affect the clotting time. The blood of patients with 2 mgms. of calcium per 100 cc. clots well. Hemorrhagic conditions are often erroneously attributed to lack of calcium and are treated by calcium salts. If the coagulation time of the blood is delayed before surgical operations, calcium salts are often given. Marriott[19] states that such a procedure is illogical, as the prolonged clotting time is not due to diminished calcium, and the administration of calcium salts will not raise the serum calcium above normal or change the clotting time.

COAGULATION AND BLEEDING TIME IN A SERIES OF TWENTY-FOUR CASES OF SENILE CATARACT.

The normal coagulation time varies between two and five minutes, while the bleeding time is slightly less. In a series of twenty-four cases of senile cataract, the coagulation time varied from three minutes and ten seconds to five minutes and fifteen seconds, with the bleeding time from two minutes and 15 seconds to four minutes and five seconds. The average was well within normal limits. It is believed, therefore, that the normal coagulation and bleeding time is further evidence that there is no deficiency of ionized calcium in the circulating blood of patients with senile cataract.

THE VITAMINES.

Szily and Edelstein[5] (1923) reported the production of lamellar cataracts in young rats nursed by a mother which was fed a diet similar to the McCollum and Simmonds diet 3143. When the litter was large they found the rats developed cataracts, whereas, when the litter was small no cataracts developed. The cataracts usually developed in the fourth week. Stepp and Friedenwald[6] (1924) attempted to repeat these experiments. They examined the eyes of rats on diet 3143, which uniformly developed rickets but not cataracts. Szily's view was that cataracts were due to a deficiency of vitamine A, excess of calcium and a deficiency of phosphorus in the diet. Stepp and Friedenwald confined animals to a diet very low in A and phosphorus but relatively rich in calcium. The animals developed both rickets and xerophthalmia but no cataracts. They conclude that diets deficient in either vitamine A or D with an excess of calcium and low phosphorus do not lead to cataracts in young rats. Friedenwald,[6] in a personal communication to McCollum, expressed the opinion that the cataracts noted by Szily were probably the result of the eyes of very young rats becoming cold from lack of protection against chilling.

Deficiency in vitamines has been found to account for peculiar disturbances of nutrition of special structures in the body. In the eyes, lack of vitamin A had been held accountable for the development of xerophthalmia in laboratory animals and in children and of keratomalacia in children. More recently, Pillat[20] has been convinced that keratomalacia occurs in adults and is due to vitamin A deficiency. In his series of cases, there was no cataract

associated with the corneal dystrophy. Other evidence may be found later to show that cataract is not dependent on vitamin A deficiency.

There has not been found any relationship between deficiency of vitamins B, C, or E and cataracts. Vitamin D is one of the factors closely associated with calcium metabolism and in this connection is particularly interesting. However, its deficiency in infancy results in rickets rather than in tetany, and it has been shown that cataract development is found in the latter rather than in the former. Furthermore, there is no evidence of lack of vitamin D in patients with senile cataract.

SUNSHINE AND ULTRAVIOLET.

The beneficial action of sunshine and ultraviolet light has been ascribed chiefly to their influence upon the calcium metabolism of the body. Burge[4] found that long exposures of ultraviolet light produced no opacity of the lens protein "in vitro" but when the latter was sensitized by calcium salts the light acted to produce opacity. However, in the human, while ultraviolet light has been held under suspicion as one of the factors in the production of cataract, it has never been proved to have any harmful effect on the normal crystalline lens. The radiant energy of the sun and artificial sunlight in the form of ultraviolet light can be absorbed by the living tissues of plants and animals under proper conditions and produce physical and chemical reactions which result in the formation of the activator substance, vitamin D. This substance makes it possible for calcium to take part in chemical reactions in the body out of all degree to the quantities present from a purely chemical standpoint. Vitamin D is an unsaturated sterol, soluble in certain fats, and is found chiefly in the fats of plants and animal origin, and of the latter the most important sources are the fats of certain fishes and the butter fat of milk.

VIOSTEROL.

The fact that a pro-vitamin, or substance that can be activated into vitamin D, is present in certain food materials was recognized almost simultaneously by Steenbock[21] and Hess,[22] and as a final result viosterol has been produced by exposing ergosterol to the action of ultraviolet light. Viosterol has been found to have all the remarkable properties of vitamin D, in concentrated form, exercising a remarkably beneficial action in cases of rickets and tetany. Thus the widespread present use of viosterol and the natural

sources of vitamin A should serve to reduce the number of cases of infantile tetany and in this manner will certainly produce, as a secondary effect, a reduction in the number of cases of lamellar and zonular cataract.

OSTEOMALACIA.

In osteomalacia, with or without hyperparathyroidism, there is a tremendous disturbance of calcium metabolism. A case of a young lady of 21 years, who for seven years had suffered from a progressive condition of osteoporosis, was observed by Turner[22] for three years at Presbyterian Hospital. She constantly excreted more calcium and phosphorus than she took in, and so she had depleted her bones of these materials. Her blood serum calcium had varied from 10.06 to 11.0 mgms. per 100 cc. Throughout the course of her illness her lenses had remained perfectly clear. Not even the slightest haze or loss of transparency was evident. A partial parathyroidectomy has not produced any tetany or any cataract.

HYPERCALCEMIA.

There are practically no cases of excess calcium in the blood except in hyperparathyroidism, some cases of osteomalacia and after excessive doses of viosterol and parathormone. In the dog,[3] the subcutaneous injection of parathormone causes a gradual rise in serum calcium for a period of fifteen hours and then a gradual fall to normal, according to dose given. If 15 mgms. of calcium per 100 cc. blood are reached, vomiting may occur. If above this, profound signs of hypercalcemia are present: vomiting, diarrhea, uneasiness. If to 20 mgms. for only a short time, recovery may occur. If maintained, it is fatal after respiratory distress and bleeding into the bowel, collapse and death. Taken by mouth the hormone has no effect. Tissue calcium increases in the fatal cases. Only in the kidney was the calcium found increased in cases still doing well. The report does not state specifically whether the calcium in the eye tissues was examined or not. There have been no cases of cataract reported in man or in animals as a consequence of hypercalcemia.

MONGOLISM.

Ormond[1] found opacities to be present in Mongolian idiocy in about 50 per cent of the cases. Jeremy[1] quoted Stoelzner as noting that mongolism is due to calcium deficiency both in the mother

and child. The author has under treatment now a child who shows unmistakable signs of mongolism and who was observed to have almost complete cataracts one week after birth. The blood serum calcium of the mother was found to be normal. There has not been any indication of any active or latent tetany in the child. The conditions of cataract and mongolism can hardly be ascribed to calcium deficiency in this case.

TOXINS.

There is apparently some uniformity of opinion that calcium is important in preventing or counteracting the effect of exogenous and endogenous toxins. This action may and probably is only secondary, acting as a base in balancing the acids that are produced with or by the toxins. In this connection it is of importance only in the general system unless one agrees with the cytotoxin theory of Roemer,[21] as an explanation of the development of cataract. One can hardly explain the production of cataract in the average patient on the toxin basis or ascribe the deposition of calcium in the cataractous lens to local toxins.

CALCIUM METABOLISM IN RELATION TO SENILE CATARACT.

For this study only the progressive types of senile cataract were selected. There are recognized the two usual forms. (1) The nuclear variety, being a pathologic form of nuclear sclerosis with hardening that may go on and involve the entire lens. The color of the lens usually changes, going from amber through shades of red to the black cataract. This color, as Burdon-Cooper[10] has shown, is due to hydrolysis of the lens protein with formation of tyrosine, which when oxidized gives the color reactions. (2) The cortical variety, starting in the senile cortex and beginning with vacuoles, water-splitting of sutures or lamellar separation, and going through the familiar stages to maturity. Then there are also (3) the posterior cortical saucer-shaped variety slowly progressive, and (4) that rarer form which begins with vacuoles and a thin layer of opacity just beneath the anterior and posterior capsules, usually in the younger old people, rapidly going on to complete opacity through diffuse clouding and swelling of the entire lens.

THE MECHANISM OF THE DEPOSITION OF CALCIUM IN SENILE CATARACT.

In order to understand the relation of the deposition of calcium to senile cataract, it is well to inquire in what form the calcium

is deposited in senile cataract and whether this is a primary or secondary process.

In normal bone formation or in fracture repair, Murray[25] has shown from his original investigations that the calcium deposition may be dependent upon a local concentration of calcium and the action of ferments, of which a phosphatase has been identified as working best in an alkalin reaction more alkalin than the blood serum. Elsewhere in the body, tissue death with granulation tissue replacement will lead to bone formation, provided the hydrogen ion concentration is right and there is a local supply of calcium available. An example of this is found undoubtedly in the abnormal bone formation in the choroid. In the lens, it is not possible to have connective tissue replace the dead or dying lens issue, and so the deposition of calcium is in some other form. Wessely and Kranz[2] state that the calcium is combined with phosphate, but Adams[2] found the tests for phosphate in cataract only very weakly positive. In this connection, however, it may be said that the test for phosphate even in bone is not conclusive. Only a small percentage of cataracts showed the deposition in the so-called spherolith form, which Wessely[2] noted in his cases of 70 years and more. It may be assumed, therefore, that these spherolíths form only at a late stage and that in the other cataracts the calcium is more evenly distributed. There is a similarity in some ways, as Adams pointed out, between the deposition in cataract and in the intima of sclerosed blood vessels where the cholesterol is markedly increased. Adams[2] could not, however, establish any intimate connection between the calcium and cholesterol in cataract such as is found in atheroma.

The presence of increased calcium in cataract is either (1) primary and an actual etiologic factor in producing lens opacity or (2) secondary to local tissue changes which exist in the lens regardless of the calcium content of the serum, aqueous or lens.

If it is primary, it is interesting to speculate on the possible mode of action in the production of the opacity. It might act (a) in reducing the permeability of the capsule. Friedenwald[26] demonstrated that in vitro, calcium has this action and has shown that under other conditions it takes only a relatively slight reduction in the permeability of the capsule to obstruct the metabolic processes of the lens sufficiently to cause cataract. (b) In sensitizing the lens protein for an abnormal action of ultraviolet light Burge[4] found that in vitro the lens protein could be so influenced by cal-

cium that ultraviolet light would act to precipitate it, but he concluded that the concentration of calcium and the amount of light necessary to produce this in vitro are not found in vivo. (c) In altering the colloidal system of the lens that precipitation of the protein occurs. Here again Adams[2] found that the conditions necessary for this reaction are not met with in the living body. Finally, it must be remembered that there is no direct evidence that the calcium of the lens is increased before cataract forms.

DEPOSITION OF CALCIUM A SECONDARY REACTION.*

One must feel, therefore, that the increase of calcium in cataract is not primary, that it is not the cause of cataract but that it is a coincident or secondary change that takes place with or follows other alterations in the lens. The deposition of calcium is probably a matter of changes in the hydrogen ion concentration and ferment activity of the lens following the production of some adsorption affinity for calcium resulting in a local concentration.

The hydrogen ion concentration and the ferment factors in the lens may be affected by the following conditions: (1) Quantitative and qualitative changes in the aqueous deleteriously affecting the nutrition and metabolism of the lens; (2) lens tissue death. These alterations in turn affect the local concentration factors of calcium inasmuch as certain byproducts of abnormal metabolism or death of lens tissue resulting from faulty metabolism have an affinity for calcium. The particular substances are the phospholipoids. These,[27] which represent about 1 per cent of the total dried substance of the normal crystalline lens, are perceptibly increased in the elderly individual to about 2 per cent, while in cataract there is as much as 13 per cent. The same holds true for the fatty acids, always at a minimum in the normal, but increased considerably in the presence of cataract. Between the element calcium, which is a bivalent cation[28] carrying a double positive charge, and the large molecule of the lipoid, which has surface ions of the opposite charge, there is an adsorption affinity which would account for the attraction of the calcium to the altered

*It is unfortunate that the article by Salit and Mattill was not quoted in the original text. In this, there is reported that the examination of five human lenses in the early stages of cataract did not show any calcium. This finding is important in ruling out an excess of calcium as being a factor in initiating the cataractous process. Reference: Salit, P. W., and Mattill, H. A. The American Journal of Physiol., Vol. XC, No. 2, October 1, 1929, p. 503.

lens. One cannot find the usual form of calcium phosphate in the lens. Adams'[2] tests for phosphate were only weakly positive. The attraction of calcium to the lens cannot be explained on the basis of the calcium as an alkali being called upon to counteract local acidity. If it performed this action, the combined molecule would probably go out through the lens capsule and be excreted. For calcium deposition, once the local concentration has been established, one needs alkalin reaction, more alkalin than the normal p H of the lens. Rapid tissue death gives a change in the p H toward the acid side. Murray has postulated that very slow tissue death, on the other hand, gives a change toward the alkalin side. This is probably what one has in the production of cataract: very slow tissue death, degeneration of the lens protein with lipoid byproducts, local concentration of calcium by adsorption to the large molecules of the lipoids, and deposition of this calcium by change in the p H toward the alkalin side.

NO TETANY OR LATENT TETANY IN CASES OF SENILE CATARACT.

In a series of twenty-four cases of senile cataract examined carefully at the Vanderbilt Clinic, no symptoms or signs of tetany or latent tetany were demonstrable. One may say that in his entire experience with senile cataract cases, no case in which one was suspicious of tetany had been seen.

THE SERUM CALCIUM LEVEL OF SENILE CATARACT CASES.

Tron,[2] using de Waard's method, investigated the serum calcium of ten persons without cataract of ages 42 to 59 years, and found an average level of 13.1 mgms. per 100 cc. In 25 cases of senile cataract the average was 12.25 mgms. Polichova[2] found the average normal 13.5 mgms., while in 14 cases of cataract it was 13.8 mgms. These values are manifestly above what is now considered to be the normal level, according to the more modern and approved technic. V. Pellathy[2] used Kramer and Tisdall's method and found the normals to range between 9.8 and 11.7, while 48 cataract cases were between 9.1 and 12.0 mgms.

Adams[2] has published the most recent report on the level of calcium in the blood of patients with senile cataract. She used an approved technic and estimated the blood serum calcium of normal and cataractous patients of similar ages. She found in the normal, 8.32 to 11.39 mgms. average 9.76 per 100 cc., while in the cataract cases the figures were 9.72 to 12.85 mgms. per

100 cc., average 11.46. She concluded, therefore, that in senile cataract the serum calcium is appreciably raised. These findings certainly contradict the theory that senile cataract is associated with a low blood calcium or parathyroid insufficiency.

STUDY OF TWO SEPARATE SERIES OF CATARACT CASES.

It is interesting to compare the results of an investigation of this subject in a series of cases at Bellevue Hospital and another series at Vanderbilt Clinic. Different assistants and technicians were employed. It cannot be said that the type of patient varied sufficiently to be of note.

TECHNIC OF ESTIMATION OF BLOOD CALCIUM.

As far as possible, sources of error were eliminated. The Kramer and Tisdall[29] method for determination of serum calcium was used in both series. The blood was taken usually in the afternoon when the patients visited the clinic.

BELLEVUE SERIES.

A total of 28 males, 15 with senile cortical cataract, 11 with senile nuclear and two with combined nuclear and cortical, ranging in age from 52 to 80 years, had serum calcium levels ranging from 8.4 to 14.3, average 11.3 mgms. per 100 cc. Total of 25 females, 14 with senile cortical, eight with nuclear, one with nuclear-cortical, and two with aphakia, ranging in age from 52 to 82, had serum calcium ranging from 9.3 to 13.7, average 10.9 mgms. per 100 cc. There was no age difference or tendency to grouping according to type of cataract. The male group was .4 mgms. higher than the female group.

It was thought at first that it would be better to reject this series because of the apparently more accurate results obtained later at Vanderbilt Clinic. However, it is similar to the series reported by Adams. If one were to select a smaller series from the 53 cases, one could prove quite a high calcium content of blood serum for cases of cataract.

The results of the two series show that for proper judgment it is necessary to have a fairly large group of cases.

STUDY OF THE SERUM CALCIUM LEVEL OF FIFTY-THREE CATARACT PATIENTS AT BELLEVUE HOSPITAL.

MALE

No.	Initials	Age	Type of Cataract	Serum Calcium
1.	F. B.	52	C.	12.3
2.	S. S.	55	C.	12.2
3.	J. R.	56	C. N.	11.2
4.	W. P.	58	C.	9.4
5.	W. C.	58	N.	10.3
6.	V. U.	60	C.	14.3
7.	C. D.	60	N.	11.2
8.	W. F.	60	N.	11.7
9.	A. C.	60	C.	12.0
10.	J. H.	61	C.	10.0
11.	J. D.	62	N.	11.2
12.	F. V.	63	C. N.	12.1
13.	S. M.	65	C.	13.8
14.	A. W.	65	C.	11.4
15.	J. C.	67	C.	13.1
16.	J. F.	67	N.	10.3
17.	R. D.	67	N.	12.0
18.	F. B.	68	N.	10.8
19.	I. K.	68	C.	11.3
20.	A. T.	68	C.	10.7
21.	A. P.	68	C.	12.5
22.	A. B.	69	C.	13.2
23.	J. F.	71	C.	8.4
24.	C. G.	73	N.	11.6
25.	J. C.	73	N.	9.5
26.	A. B.	75	N.	9.8
27.	J. O.	77	N.	12.4
28.	F. O.	80	C.	10.4

Average 11.3 mgms. per 100 cc.

FEMALE

No.	Initials	Age	Type of Cataract	Serum Calcium
1.	E. M.	52	N.	12.9
2.	M. B.	54	C.	11.0
3.	C. M.	55	APH	8.7
4.	C. S.	58	C.	11.8
5.	B. L.	58	C.	12.5
6.	D. E.	58	C.	10.3
7.	A. B.	60	C.	9.8
8.	F. D.	60	C.	10.8
9.	M. J.	60	N.	10.6
10.	B. B.	60	C.	13.7
11.	C. T.	62	C.	9.3
12.	M. C.	64	N.	10.0
13.	C. A.	64	N.	10.6
14.	E. M.	65	C.	13.7
15.	M. H.	65	APH	10.5
16.	R. B.	66	N.	10.8
17.	S. S.	66	C.	11.2
18.	E. D.	68	N.	9.3
19.	P. M.	69	N.	10.6
20.	S. S.	70	C.	9.5
21.	M. S.	72	C.	12.5
22.	E. R.	73	C.	10.2
23.	K. K.	78	N.	10.5
24.	M. G.	81	C.	10.1
25.	A. K.	82	C. N.	10.6

Average 10.9 mgms. per 100 cc.

Legend: C, cortical; N, nuclear; C.N., corticonuclear; APH, aphakia.

VANDERBILT CLINIC SERIES OF CONTROL CASES WITHOUT CATARACT.

A total of eighteen cases without cataract in whom the serum calcium level had been estimated was used for purpose of comparison. There were 14 males and 4 females, ranging in age from 32 to 67, with various eye disorders. The serum calcium values ranged from 9.7 to 11.4, average 10.6 mgms. per 100 cc. This entire series cannot be regarded as normal cases such as Adams used in her control series, but eight of the cases can be so looked upon. The average for these eight cases is the same as for the 18 total and for the 10 pathologic cases.

STUDY OF BLOOD SERUM CALCIUM OF EIGHTEEN CONTROL CASES.

Case No.	Initials	Age	Sex	Vision R. E.	Vision L. E.	Serum Calcium	Remarks
1.	M. M.	32	M.	10/200	10/200	10.3	Lues
2.	J. M.	32	M.	20/70	20/100	11.7	Dystrophy of cornea
3.	M. K.	39	M.	Nil	20/20	10.5	Embolism of cent. artery
4.	H. F.	39	M.	20/20—	20/30+	10.3	Uveitis
5.	M. K.	45	M.	20/40	20/20	11.4	Optic atrophy O. D.
6.	H. F.	47	M.	20/20	20/40	10.7	Lues
7.	L. T.	49	M.	3/200	Nil	10.4	Optic atrophy : Lues
8.	S. L.	52	M.	20/40	20/40	11.0	Control
9.	P. D.	55	M.	20/40	20/100	10.2	Retinitis pigmentosa
10.	F. J.	56	M.	20/20	20/30	10.3	Control
11.	M. P.	58	M.	20/20	20/20	9.7	Control
12.	I. K.	60	M.	20/20	20/40+1	11.0	Control
13.	I. M.	62	M.	5/200	2/200	10.8	Glaucoma. Atrophy
14.	A. G.	69	M.	20/40	20/50	10.8	Control
15.	B. E.	49	F.	20/20	20/20	11.2	Control
16.	L. B.	57	F.	20/40	20/40	10.3	Control
17.	E. M.	67	F.	20/70	20/70	9.7	Glaucoma
18.	A. G.	67	F.	20/20	20/40	10.7	Control

Average 10.6 mgms. calcium per 100 cc.

This series is small but it need not be made larger, as the normal figures for all types and ages have been worked out by many observers as between 9 and 11 mgms. per 100 cc. of blood serum.

STUDY OF BLOOD SERUM CALCIUM LEVEL IN CASES OF CATARACTS AT VANDERBILT CLINIC.

A total of 33 males and 50 females with cataract were studied as to their age, type of cataract, vision with correcting glasses and complicating factors in relation to their blood serum calcium level.

The males ranged in age from 25 to 82 years, the females from 12 to 82 years. No relation between age and serum calcium level could be found. The average for the two sexes was only .05 mgms. per 100 cc. apart, 10.29 for the males and 10.34 mgms. per 100 cc. for the females.

The series was selected with the aid of the slit-lamp and microscope. A few cases of cataract in younger persons were included. A predominance of cortical cataracts was found, 52 in number, and the average serum calcium level was found to be 10.36 mgms. per 100 cc. Nine nuclear cataracts gave an average of 10.22. Eight corticonuclear cases were also found to have an average of 10.22. The posterior cortical variety were nine in number and averaged 10.01; the three lamellar cataracts were 10.2 while the equatorial wedges and coronary types were one each with 10.7 and

10.5, respectively. There was no relation of the calcium level to the visual quotient in any of the various types.

Diabetes was the most common complicating or etiologic factor found. Only those cases with sugar in the urine were noted in this cataract series. It does not include all those which are discoverable by the glucose tolerance test. The average calcium level for 12 cases of diabetes was 10.58. For the four cases of glaucoma, 10.27; for three cases of uveitis, 10.53; for four cases of otosclerosis, 9.67; while for those with kidney and vascular disease it was found to be 10.65 mgms. per 100 cc.

Manifestly there is no significant relationship in the matter of the serum calcium level, and the factors of age, sex, type of cataract, visual quotient, complicating or incidental disease. The average and the greater percentage of the cases are well within the normal limits.

There were only eight above the upper limit of 11 mgms. and only one below the lower limit of 9 mgms. per 100 cc. There were 24 between 9 and 10 mgms., with an average of 9.7; 47 were between 10 and 11 mgms., with an average of 10.46 mgms. per 100 cc. As far as can be judged from the serum calcium levels of these patients, there is no deficiency of calcium in cases of senile cataract. The tendency is to the upper normal limits. Therefore, as judged by the serum calcium level, there is no deficiency of parathyroid secretion in elderly people with cataract but actually normal levels in the greater percentage with a tendency to a very mild hypercalcemia in a few.

STUDY OF BLOOD SERUM CALCIUM IN CASES OF CATARACT.

THIRTY-THREE MALES.

Case No.	Initials	Age	Vision R. E.	Vision L. E.	Type of Cataract	Serum Calcium	Remarks
1.	R. G.	25	20/50	20/100	Lamellar	9.8	
2.	S. K.	29	20/70	20/70	Post-cortical	10.6	Retinitis pigmentosa
3.	J. McV.	30	20/20+	H. M.	Post-cortical	10.0	Uveitis. Glaucoma
4.	C. L.	35	20/70	20/70	Lamellar	10.0	
5.	M. B.	43	20/40	H. M.	Equatorial	10.7	Lues. Atrophy
				SENILE CATARACT.			
6.	P. D.	45	20/40	20/20	Cortical	10.2	
7.	M. O.	49	H. M.	20/70	Cortical-nuclear	10.5	Diabetes
8.	S. G.	50	20/40	20/70	Post-cortical	10.6	Old iritis
9.	H. B.	51	20/200	20/60	Cortical	10.4	
10.	S. S.	55	20/40	20/70	Cortical-nuclear	10.4	
11.	S. S.	57	20/25	20/25	Cortical	9.9	
12.	L. B.	57	H. M.	L. P.	Nuclear-cortical	10.3	Diabetes
13.	J. F.	57	20/70	L. P.	Post-cortical	8.9	Otosclerosis
14.	L. R.	59	20/200	20/40	Nuclear	10.9	
15.	A. Y.	59	20/30	20/30	Cortical	11.1	
16.	M. A.	61	20/40	20/200	Cortical-nuclear	10.3	
17.	M. A.	61	20/70	20/200	Cortical	10.4	
18.	T. W.	66	20/40	20/30	Cortical	9.8	
19.	B. S.	67	20/200	H. M.	Cortical	10.0	
20.	H. D.	68	L. P.	20/100	Cortical	9.6	
21.	H. S.	68	20/200	20/100	Cortical	10.7	
22.	M. R.	68	20/30+	20/30	Cortical	10.3	
23.	A. R.	69	L. P.	20/50	Post-cortical	10.6	Trauma. Contusion
24.	P. G.	69	20/30	20/40	Cortical	10.4	
25.	S. P.	70	20/40	20/70	Cortical	10.3	
26.	J. M.	70	H. M.	20/70	Nuclear-cortical	11.0	Choroiditis
27.	P. T.	72	20/40	20/40	Cortical	10.4	Aphakia. Diabetes
28.	A. L.	72	20/70	20/70	Cortical	11.1	Diabetes. Retinitis
29.	S. S.	78	20/70	20/50	Cortical	9.9	
30.	F. O.	78	20/200	10/200	Cortical	11.0	Macular degeneration
31.	W. S.	81	L. P.	H. M.	Post-cortical	9.7	Marked glaucoma
32.	C. H.	82	20/70	20/50	Nuclear	10.3	Otosclerosis
33.	D. M.	82	H. M.	H. M.	Nuclear-cortical	9.6	
					Average	10.29	

STUDY OF BLOOD SERUM CALCIUM IN CASES OF CATARACT.

FIFTY FEMALES.

Case No.	Initials	Age	Vision R. E.	Vision L. E.	Type of Cataract	Serum Calcium	Remarks
1.	L. K.	12	20/40—1	20/40—2	Lamellar	10.8	
2.	E. A.	37	20/20	20/20	Coronary	10.5	
3.	A. S.	44	20/30	15/200	Nuclear	9.9	Hypertension. Retinitis
4.	L. S.	47	20/200	20/40	Cortical	10.8	Glaucoma
5.	E. G.	47	20/100	Nil	Post-cortical	10.7	Glaucoma
			SENILE CATARACT.				
6.	S. S.	50	20/40	20/40	Cortical	11.5	Hypertension
7.	M. V.	52	20/70	20/50	Cortical	10.0	Diabetes. Retinitis
8.	M. T.	54	20/40	20/40	Cortical	10.1	
9.	A. B.	54	20/40	20/40	Cortical	10.6	
10.	M. R.	55	20/20—	20/20—	Cortical	9.9	
11.	A. L.	55	L. P.	10/200	Cortical	10.5	Diabetes
12.	S. M.	55	20/40	20/200	Cortical	11.2	Hypertension. Retinitis
13.	A. D.	55	20/40	20/20	Cortical	10.8	
14.	M. P.	56	L. P.	20/70	Cortical	10.8	
15.	L. R.	56	20/30	20/30	Cortical	10.7	
16.	P. C.	57	10/200	20/70	Nuclear	10.6	High myopia
17.	D. F.	57	20/70	20/200	Post-cortical	9.3	Retinitis pigmentosa
18.	M. K.	58	20/70	20/70	Cortical	10.3	Diabetes
19.	A. S.	58	H. M.	20/200	Cortical	10.6	Diabetes
20.	L. S.	58	20/30—	20/30—	Post-cortical	9.7	
21.	B. D.	59	20/20—	20/50	Cortical	9.5	
22.	S. S.	60	20/200	20/200	Nuclear	9.9	Glaucoma

23.	C. S.	62	20/30	20/40	Nuclear	9.8	Myocarditis
24.	D. B.	63	20/70	20/70	Nuclear	9.9	
25.	B. B.	63	20/30—	H. M.	Cortical	10.4	
26.	S. W.	64	20/40	20/40	Cortical	11.6	
27.	C. H.	64	5/200	20/100	Cortical	10.7	Diabetes. Nephritis
28.	K. B.	64	20/30	20/20	Cortical	9.5	
29.	R. E.	64	20/40	20/40—	Cortical	10.5	Diabetes
30.	R. G.	65	20/200	10/200	Cortical-nuclear	11.4	Diabetes
31.	R. F.	65	20/100	20/70	Cortical	10.8	Hypertension. Retinitis
32.	H. T.	65	20/50+3	20/50+3	Cortical	9.8	
33.	E. H.	67	20/40	20/40	Cortical	10.6	
34.	M. P.	67	20/50	L. P.	Cortical	11.2	
35.	B. B.	67	20/30	20/30	Cortical	10.7	Diabetes
36.	D. B.	68	20/50	20/40	Cortical	10.0	
37.	E. J.	68	20/70	20/100	Cortical	10.0	
38.	L. B.	69	20/30	20/30	Cortical	9.2	Otosclerosis
39.	P. H.	69	20/30	20/40	Cortical	9.8	
40.	E. S.	69	H. M.	H. M.	Cortical-nuclear	10.3	
41.	F. G.	69	H. M.	H. M.	Cortical	9.9	
42.	J. D.	69	20/40	20/40	Cortical	9.6	
43.	C. U.	69	20/70	20/70	Cortical	9.8	
44.	D. F.	70	20/20—	20/20—	Nuclear	9.5	
45.	E. M.	70	20/70	20/30	Cortical	10.0	
46.	C. H.	74	20/200	20/50	Nuclear	10.4	Hemorrhagic retinitis
47.	M. D.	75	10/200	10/200	Nuclear-cortical	9.4	
48.	M. U.	78	20/40	20/70	Cortical	11.2	
49.	M. K.	78	20/200	20/200	Nuclear	11.0	
50.	A. K.	82	20/30	20/50	Cortical	10.3	Otosclerosis
					Average	10.3	

SPINAL FLUID CALCIUM IN CASES OF SENILE CATARACT.

Because of the similarity in formation and composition of the aqueous and spinal fluid, it was decided to study the level of the calcium of the spinal fluid in cases of senile cataract at Bellevue Hospital.

Case No.	Initials	Sex	Age	Type of Cataract	Blood Serum Calcium Mgms. per 100 cc.	Spinal Fluid Calcium Mgms. per 100 cc.
1.	C. S.	F.	58	Cortical	11.8	5.9
2.	M. S.	F.	72	Cortical-glaucoma	12.5	7.5
3.	W. F.	M.	60	Coronary	11.7	8.5
4.	C. D.	M.	61	Nuclear	11.2	6.7
5.	J. R.	M.	56	Nuclear	11.2	8.7
6.	J. D.	M.	62	Nuclear-glaucoma	11.2	6.8
7.	A. K.	F.	82	Cortical-nuclear	10.6	7.5
8.	F. O.	M.	80	Cortical	10.4	6.8
				Average.....	11.3	7.3

These cases all had serum calcium at or slightly above the upper limits of normal. The spinal fluid calcium figures reflected the same condition. If one may judge from this, there is no deficiency of calcium in the aqueous in these cases but really a slight excess.

In this connection, one is struck by the possible significance of Burge's work. Lens protein, while not precipitated by ultraviolet light alone, is precipitated by ultraviolet light in the presence of an excess of electrolytes. The possibility of the excess calcium found in a certain number of the cases of the series of senile cataract being a primary factor in the production of the opacities will be discussed later.

A STUDY OF THE ENDOGENOUS CALCIUM METABOLISM OF PATIENTS WITH CATARACT.*

It was realized that the serum calcium level did not represent the flow of the element, either into the tissues or into the excreta, nor the relation of calcium intake to output. In children and adolescents the need for development of bony structure requires a great disproportion between intake and output, with a flow of calcium into and storage in the bony tissues. In adults and in

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the elderly there is need for a balance between intake and output. There is normal metabolic exchange of calcium in their bony tissues and a storage in the fine bony trabeculæ to meet emergencies when available base is called from the alkali reserve. Knowing that patients with cataract had normal serum calcium level with a tendency in 66 per cent to slightly higher normal limits, it was desirable to know whether these latter had excess parathyroid secretion, excess acid production from dietary error or from tissue breakdown and whether they were storing or excreting the excess. If they were storing it, was it going into the normal storehouse, the bony trabeculæ or into tissues such as the lens? If the latter, could agents which favored excretion withdraw it from the lens and restore part of the transparency of the lens tissue?

COMPLETE CONTROL NECESSARY.

The study of the relation of the intake and output of calcium in patients with cataract was first undertaken in cases which were being operated upon for cataract. They were in the general eye ward at the Presbyterian Hospital. It was soon realized that conditions while ideal from the surgical standpoint, did not offer full control over diet and the care of their specimens. The bowels were necessarily irregular during the period of their being in bed after the removal of their cataracts, so that the estimation of the fecal content of calcium when specimens were obtained after catharsis did not give the proper relation between intake and output. The urinary output was more constant, but that alone does not give the proper basis for judgment of the situation. Sixteen cases were examined under these conditions, and necessarily no conclusions can be drawn from the data obtained. It was, therefore, with proper appreciation that the facilities of the male metabolism ward in Presbyterian Hospital were used when offered for the continuation of the study under controlled conditions.

STUDIES IN METABOLISM WARD.

Six patients were selected for this study. Complete histories, physical examinations, blood, metabolic and functional tests were performed before they were placed in the calcium metabolism ward. Here the intake and output in the excreta were carefully calculated. One must realize the laborious nature of such a study and that it can be carried out only through the intelligent and interested cooperation of dietitians, nurses, attendants and

technicians. All the factors bearing on the calcium metabolism were controlled. The patients were weighed regularly. The amounts of carbohydrate, fat and protein in the diet were ordered and the total calories estimated. The fluid intake and output was measured. The acid and base residue equivalent of the diet was calculated and for the preliminary tests was kept as near the neutral point as possible. Only males were selected because of the greater ease of obtaining the unmixed and complete specimens.

Case No. 1.—A. Y., white male, 60 years of age, No. 229530, with incipient senile cortical cataracts in the form of lamellar separation in the inferionasal quadrant of each lens. Vision, O. D. cc. 20/25; O. S. cc. 20/25. Physical examination, chronic pharyngitis, arthritis, cardiovascular negative. Glucose tolerance normal. Blood creatinin, 1.3. Cholesterin, 0.21. Blood carbon dioxide combining power, 60 per cent. Kidney function tests normal. On June 19, 1930, blood serum calcium, 11.1 mgms. per 100 cc. After the parathormone injections on June 27, 1930, it was 13.0 mgm.

Date June, 1930	Calcium Intake Mgms.	Calcium in Urine	Calcium in Stool	Total Calcium Excreted	Calcium Exchange	Parathormone	Remarks
17-18	654	247	647	894	—240	0	Neutral diet for all 3 periods.
18-19	598	220	327	547	+51	0	
19-20	488	184	396	580	—92	0	
20-21	850	206	420	626	+224	0	Average negative balance —6.0 mgms.
21-22	566	180	362	542	+24	0	
22-23	671	191	463	654	+17	30 units	Average negative balance on parathormone —163 mgms.
23-24	512	185	943	1128	—616	30 "	
24-25	—?	282	738	1020	—?	30 "	
25-26	745	190	530	720	+25	30 "	
26-27	532	184	430	614	—82	30 "	
27-28	841	210	475	685	+156	0	After parathormone, average daily negative balance —88 mgms.
28-29	576	167	407	574	+2	0	
29-30	612	195	840	1035	—423	0	

This patient had a perfectly normal calcium balance on a neutral diet as determined in the first five-day period. He excreted only 0.6 mgms. on the average per day, more than he took in. The action of parathormone* is evident in the increased excretion of calcium during the five days of the daily injections of 30 units, by the negative balance, excreting an average of 162 mgms. more than he ingested and also for the three days following, a further average negative balance of 88 mgms.

*The parathormone used in these investigations was generously supplied by Eli Lilly Company.

Case No. 2.—A. P., a white male, 69 years of age, No. 240132, with senile corticonuclear cataracts in the form of hazy amber colored nucleus with lamellar separation in the cortex. Vision, O. D. cc. 20/30; O. S. cc. 20/50. Physical examination showed moderate obesity, dental caries, cardiac hypertrophy, hypertension. Kidney function tests showed moderate impairment. Glucose tolerance test showed normal fasting blood sugar but poor utilization. The serum calcium was 10.0 mgms. per 100 cc. In contrast to case No 1, the bowel movements of this patient were irregular, so that it was difficult to compare the first period with the second, as far as the excretion of calcium by bowel was concerned. The urinary excretion, however, was typical. In the first five control days there was an average urinary excretion of 171 mgms. per day. He was then given 30 units of parathormone on the fifth day and for the following six days 50 units per day. The result was typical as far as the increased urinary excretion was concerned. The average daily output was 216 mgms., an increase of 45 mgms. per day. When the stool calcium was figured in also the patient was found to be in negative balance all through the test. The serum calcium was found to be 10.0 mgm. per 100 cc. on June 23, 1930, before parathormone and 10.62 mgms. per 100 cc. after 50 units of parathormone on June 26, 1930, while it was 10.8 mgms. after 50 units on June 28, 1930.

Case No. 3.—M. W., white male, aged 53 years, No. 204743, with corticonuclear cataracts showing nuclear haze and lamellar separation. Vision, O. D. cc. 20/40; O. S. cc. 20/50. Physical examination showed chronic constipation, sinusitis and pleuritis. The kidney function tests were normal. The glucose tolerance test was within normal limits. There was no evidence of acidosis.

Date July, 1930	Calcium Intake Mgms.	Calcium in Urine	Calcium in Stool	Total Calcium Excreted	Calcium Exchange	Acid in Diet	Alkali in Diet
1- 2	530	210	266	476	+54	43.6	44.7
2- 3	808	141	505	646	+162	44.8	42.4
3- 4	529	162	420	582	—53	44.3	45.0
4- 5	517	180	495	675	—158	42.1	43.7
5- 6	538	166	500	666	—128	44.1	45.2
6- 7	826	180	352	532	+294	47.1	48.2
7- 8	529	165	600	765	—236	44.3	43.7
8- 9	817	230	470	700	+117	47.1	48.2
9-10	580	248	370	618	—38	65.8	37.7
10-11	828	250	374	624	+204	58.2	41.8
11-12	576	250	1080	1330	—754	65.4	40.4
12-13	608	228	255	483	+125	57.6	42.4
13-14	621	220	735	955	—334	48.7	31.5
14-15	570	316	630	946	—376	58.5	38.0

The average urinary excretion of calcium was 179 mgms. per day on the neutral diet, containing on the average 0.7 alkali residue per day.* While on acid diet, where the acid residue was equivalent to 20.4 cc. of normal solution per day, the average daily calcium urinary output increased to 252 mgms. For the control period of eight days he was in weak positive balance, storing an average of 8 mgms. of calcium per day, while from the effect of the acid diet he went into definite negative balance, excreting 196 mgms. per day more than he took in. The serum calcium in this case was 9.2 mgms. per 100 cc. on February 24, 1930, and then again found to be 11.6 mgms. per 100 cc. serum on July 28, 1930.

Case No. 4.—S. K., male, aged 47 years, No. 261174, with that rapidly progressive type of anterior and posterior subcapsular cataracts. The vision was, O. D. cc. 20/70; O. S. cc. 20/40. His general physical condition showed vascular sclerosis abnormal for his years and some cardiac hypertrophy. He was moderately obese. This and his general physique suggested hypothyroidism but his basal metabolic rate was plus 8. His glucose tolerance was normal. His kidney function was good. His blood urea was 28, uric acid 4.5, creatinin 1.8, glucose 88. His carbon dioxide combining power was 54.1 per cent. Blood serum calcium, 12.2 mgms. per 100 cc. His diet while under observation was slightly acid in residue.

Date July, 1930	Calcium Intake Mgms.	Calcium in Urine	Calcium in Stool	Total Calcium Excreted	Calcium Exchange	Acid in Diet	Alkali in Diet
17-18	586	274	113	387	+199	49.0	47.1
18-19	425	194	48	242	+183	40.4	40.4
19-20	605	124	311	435	+170	36.0	28.4
20-21	809	226	970	1196	—387	47.1	44.6
21-22	641	232	405	637	+4	40.3	40.6
22-23	777	457	125	562	+195	41.6	40.2
23-24	826	242	377	619	+207	47.1	48.2
24-25	570	240	1350	1590	—1020	57.6	40.4

This patient apparently was storing calcium for the first three days, but when his bowel moved completely on the fourth day the stool contained the extra calcium which either had not been absorbed or had been retained in the intestine after excretion so that he was in an average positive balance of 41 mgms. In the latter four days he was again irregular in bowel movements, but

*The values for acid and alkali in the diet are in terms of cc. of normal solution. A normal solution is one which contains in each litre enough of the reagent to unite with or replace one gram of hydrogen.

again the stool on the eighth day contained a large amount of calcium, and for the period he was in negative balance, excreting 154 mgms. per day more calcium than he ingested, either as a result of the slight acid residue in his diet or from his own production of acid. For the total period of eight days he was in an average negative balance of 56 mgms. per day. The diet contianed an average daily acid residue of 3.7.

Case No. 5.—W. Z., a white male, 47 years of age, No. 196364, with posterior cortical cataracts complicating retinitis pigmentosa. The vision was, O. D. cc. 20/200; O. S. cc. 20/100. The general physical condition showed a spastic paraplegia due to a spinal cord lesion. The cause of this was not known. The kidney function tests showed normal results. The glucose tolerance was within normal limits. His blood urea was 32 mgms. Uric acid, 3 mgms. Creatinin, 1.5 mgms. His carbon dioxide combining power was 51.3 per cent. His serum calcium was 10.1 mgms. per 100 cc. His calcium exchange was as follows:

Date July, 1930	Calcium Intake Mgms.	Calcium in Urine	Calcium in Stool	Total Calcium Excreted	Calcium Exchange	Acid in Diet	Alkali in Diet	Remarks
19-20	554	281	645	926	—372	30.3	35.3	1st Period. Diet contained an alkali excess of 3.6 cc. of normal sol. per day. Average positive calcium bal. +26.0 mgms.
20-21	790	337	600	937	—147	41.6	48.2	
21-22	641	408	230	638	+3	40.3	40.6	
22-23	777	202	324	526	+251	41.6	48.2	
23-24	826	220	208	428	+398	47.1	48.2	
24-25	579	340	931	1271	—692	57.6	40.4	2nd Period. Diet contained an acid excess of 18.4 cc. of normal solution. Average daily negative calcium balance —213 mgms.
25-26	635	361	324	685	—50	49.4	31.5	
26-27	591	405	...	405	+186	65.8	40.7	
27-28	606	372	348	720	—114	48.7	28.5	
28-29	691	370	579	949	—258	52.1	45.7	
29-30	569	450	112	562	+7	52.2	35.1	
30-31	591	568	588	1156	—565	65.8	40.7	
31- 1	570	420	198	618	—48	57.6	40.4	3rd Period. Diet contained an acid excess of 18.7 cc. of normal sol. 50 units of parathormone per day. Average negative calcium bal. —163 mgms.
1- 2	635	478	472	950	—315	49.4	31.5	
2- 3	591	382	620	1002	—411	65.8	40.7	
3- 4	620	230	371	601	+19	48.7	33.5	
4- 5	569	302	319	621	—52	52.2	35.1	

In the first five days, while on a slightly alkalin diet, containing an alkali residue of 3.6 cc. of normal solution per day, the patient stored an average of 26 mgms. of calcium per day, being in weak positive balance. In the second period of seven days, when his diet contained an acid residue averaging 18.4 cc. of

normal solution per day, he excreted on the average 212 mgms. of calcium per day more than he ingested. The increased excretion was chiefly by the kidneys, the stool calcium remaining practically the same. In the third period the diet contained an acid residue of 18.7 cc. of normal solution per day. He received also 50 units of parathormone per day for five days. The patient was in negative balance, excreting an excess of 163 mgms. of calcium per day.

Case No. 6.—E. B., a white male, 70 years of age, No. 256589, with senile nuclear cataract complicating glaucoma, was selected as a control case. The physical examination showed general arteriosclerosis and hypertension. The kidney function tests showed moderate decrease in efficiency. The glucose tolerance test showed very slight decrease in utilization. Blood urea, 40; uric acid, 2.8; creatinin, 1.5. The carbon dioxide combining power of the blood was 53.2 per cent. Serum calcium was 13 mgms. per 100 cc. on July 3, 1930, and 13.1 mgms. on August 4, 1930.

Date July, 1930	Calcium Intake Mgms.	Calcium in Urine	Calcium in Stool	Total Calcium Excreted	Calcium Exchange	Acid in Diet	Alkali in Diet
4- 5	529	108	390	498	+31	40.2	41.3
5- 6	603	206	...	206	+397	47.1	46.2
6- 7	775	103	465	568	+207	43.1	44.2
7- 8	501	144	...	144	+357	44.3	43.7
8- 9	366	157	520	677	—311	34.7	35.0
9-10	371	175	354	529	—158	35.1	36.1
10-11	525	152	240	392	+133	41.0	40.4
11-12	521	106	557	663	—142	42.1	40.6
12-13	462	190	229	419	+43	46.4	37.6
13-14	479	122	286	408	+71	51.2	42.5
14-15	496	258	306	564	—68	37.8	29.6

In the first period of six days the diet was approximately neutral, containing 0.3 alkali residue. The patient stored on the average 87 mgms. of calcium per day and was in definite positive balance.

This patient ate very little and rejected the food which was intended to produce the acid residue in his diet. However, in the second period, there was an average acid residue equivalent of 5.7 cc. of normal solution per day. The average daily urinary excretion of calcium was 149 mgms. per day during the control period on neutral diet, while it was increased to 165 mgms. per day on a slightly acid diet. The weakly acid residue diet caused the definite positive calcium balance of 87 mgms. per day to be reduced to a very weak positive balance of 8 mgms. per day.

Summary of Results.

THE EFFECT OF APPROXIMATELY NEUTRAL DIET ON CALCIUM BALANCE.

Case No.	Av. Calcium Intake per day	Av. Calcium in Urine per day	Av. Calcium in Stool per day	Av. Cal. Total excreted	Av. Calcium exch. per day
1.	631	207	430	637	—6.0
3.	637	178	451	629	+8.0
5.	717	290	401	691	+26.0
6.	524	149	288	437	+87.0

THE EFFECT OF ACID RESIDUE IN THE DIET.

Case No.	Av. Daily Intake of Calcium	Av. Calcium in Urine	Av. Calc. in Stool	Av. Total Calcium	Av. Calcium Exchange	Av. Acid Residue
3.	630	252	574	826	—196	18.7
4.	655	249	462	711	—56	3.7
5.	608	410	411	821	—213	18.4
6.	496	165	323	488	+8	5.5

THE EFFECT OF PARATHORMONE.

Case No.	Av. Calc. Intake	Av. Calc. Urine	Av. Calc. Stool	Av. Total Calcium	Av. Calc. Exchange	Parathormone Daily Units	Parathormone Number of Days
1.	615	187	591	778	—163	30	5

THE EFFECT OF PARATHORMONE PLUS AVERAGE ACID RESIDUE 18.5 PER DAY.

Case No.	Av. Calc. Intake	Av. Calc. Urine	Av. Calc. Stool	Av. Total Calcium	Av. Calc. Exchange	Parathormone Daily Units	Parathormone Number of Days
5.	595	362	396	758	—163	50	5

THE EFFECT OF PARATHORMONE ON URINE EXCRETION.

CASE NO. 2.

	Average Calcium Intake	Average Calcium in Urine	Parathormone
1st period (5 days)	617	171	30 units on last day
2nd period (6 days)	674	216	50 units each day
3rd period (3 days)	740	217	None

ANALYSIS OF SUMMARY OF RESULTS.

1. Four patients with senile cataract were placed on neutral or slightly alkalin residue diet and their endogenous calcium metabolism studied. Two were practically in perfect balance when intake and excretion of calcium was compared. The other two were in positive balance, storing a small amount of calcium, most probably because of the slight alkalin residue of their diet.

2. Four patients were placed on acid residue diet. Two of the diets contained a small excess of acid and the other two contained considerably more acid. Three of the patients had been studied while in neutral or slighty alkalin diet. The response to the acid diet was typical. They excreted considerably more calcium than they did while on the neutral diet. In cases No. 3 and No. 6 both the urine and stool calcium was increased, but

the urine calcium was increased more than the stool calcium. In case No. 5, chiefly the urine excretion was increased. This is the typical effect of acid on the calcium metabolism. In case No. 6 the patient was still in positive balance, but to a much less degree than when on neutral diet. In cases No. 3 and No. 5, the acid residue was greater than in No. 4 and No. 6, and the excess excretion of calcium was correspondingly greater.

3. Parathormone produced increased excretion of calcium. However, in case No. 1 this increase is not typical as it is an increase of the stool calcium and not of the urine calcium. It is interesting to note that case No. 5, while kept on an acid residue diet equivalent to that of a previous test period, did not excrete additional calcium in response to parathormone beyond that which was called forth by the acid residue diet. In case No. 2, the calcium excretion in the urine was increased by parathormone.

COMMENTS ON THE ANALYSIS.

1. The important issues to be considered are those which are practical from a clinical standpoint. Do these senile cataract cases have any need for calcium therapy? As far as can be judged from this small series of cases investigated thoroughly, one can say that there is no calcium deficiency in these patients, and that they do not require calcium administration beyond that which can be obtained in a normal diet. Their diet should be neutral or slightly alkalin in residue to keep them in calcium balance and to avoid excess drain upon the calcium reserves. It is far wiser to correct the diet if need be, than to allow the patient to continue on an acid residue diet and administer calcium salts to make up for the excessive excretion of calcium. There was no evidence of an excessive endogenous production of acid in these patients.

2. Do these cases of senile cataract need parathyroid extract? Here the answer must be in the negative. The best regime for elderly patients is one which keeps them in calcium balance or near there as they vary each day. The cases investigated were near the balance point when on neutral or slightly alkalin diet. There is no need for parathormone, as the excess calcium excreted comes out of the bones and not out of the cataracts. There is no need for raising the serum calcium with parathormone, as in the greater percentage of these cases it is high enough.

3. Are these patients utilizing their calcium properly? The answer is in the positive, for they show typical responses to neutral, alkalin and acid residue diets and to parathyroid extract.

THE USE OF PARATHYROID EXTRACT IN SENILE CATARACT CASES.

Before it was learned that the calcium metabolism of patients with senile cataract was within normal limits and that they did not need parathyroid extract treatment, it was thought that possibly they might benefit from the use of the latter. There had come to the author's attention certain unpublished reports of the use of various extracts with more or less success. As calcium enters into the mechanism of so many physiologic processes, Collip thought it possible that clinical research would later indicate that it was desirable under definite circumstances to keep certain patients in a state of mild hypercalcemia. Besides there were other possibilities: (1) That although the greater percentage of patients with cataract had more than sufficient calcium in their blood serum, the amount which was diffusing into the aqueous was insufficient to keep up a proper balance; (2) that there was really an excess of calcium in the aqueous, vitreous and lens in the early stages of cataract and the use of the parathyroid extract would help remove some of the excess and favor the excretion of it.

These matters have been cleared up greatly in the progress of the study, and the answers to these questions have been given in the preceding pages of this paper. However, it was important to know the action of parathyroid extract in cases of cataract, and so the most potent and the only reliable form, parathormone, was administered to selected cases.

EFFECT OF DAILY INJECTIONS OF PARATHORMONE ON CATARACT CASES.

Six cases of cataract were given five daily subcutaneous injections of parathormone in 1.5 cc. doses (30 units).

Most of these were fairly well advanced cases of cataract, cases in which one might suspect from the appearance of the cataracts that there were calcium deposits in the lens. Not any of them showed any improvement or absorption of the cataractous material.

One patient, A. P., male, 60 years of age, with corticonuclear cataracts, with serum calcium of 10.0 mgms. per 100 cc., was given 30 units on one day and then 50 units of parathormone daily for six days without any absorption of the cataract. The serum calcium rose to 10.62 and 10.8 after the injections of parathormone.

EFFECT OF DAILY INJECTIONS OF PARATHORMONE ON CATARACT CASES.

Case No.	Initials	Sex	Age	Type of Cataract	Vision R. E.	Vision L. E.	Serum Cal. Before Inj.	Serum Cal. After Inj.	Remarks
224316	S. K.	M.	30	Post-cortical	20/70	20/70	10.5	10.1	Ret. pigmentosa
241430	H. D.	M.	68	Cortical	L. P.	20/200	9.6	9.9	Extraction O. D.
70725	C. U.	F.	69	Cortical	20/50	20/40	9.8	10.6	Hem. ret. glauc.
86156	M. K.	F.	58	Cortical	H. M.	L. P.	10.0	10.3	Diab. extract
225267	A. S.	F.	58	Cortical	H. M.	20/70	10.1	10.3	Extraction O. D.
229153	N. P.	F.	56	Cortical	L. P.	20/70	10.8	11.5	Extraction O. D.

EFFECT OF INJECTIONS OF PARATHORMONE OVER A PERIOD OF ONE MONTH.

Case No.	Initials	Sex	Age	Type of Cataract	Vision R. E.	Vision L. E.	Serum Calcium Before	Serum Calcium After	Units of Parathormone in 1 mo.	Remarks
256661	E. J.	F.	68	Cort. nuclear	20/70	20/100	10.0	8.9	180	
258817	S. F.	F.	68	Cortical	20/40	20/50	10.6	12.1	180	
258835	P. D.	F.	45	Post-cortical	20/50	20/20	...	12.1	260	
218834	A. H.	F.	54	Cortical	20/40	20/40	10.6	9.0	240	Arrested pul. T. B.
196661	S. S.	F.	50	Cortical	20/40	20/40	11.5	10.5	290	Hypertension

EFFECT OF INJECTIONS OF PARATHORMONE OVER A PERIOD OF ONE MONTH.

Five cases of incipient and immature cataract were selected and parathormone administered in one cc. doses (20 units) subcutaneously.

On reexamination after the injections were stopped, no changes were noted in the lens opacities. The vision and refraction remained the same. It is interesting to note that three of the cases actually showed a diminution in the serum calcium taken two days after the last injection. One cannot expect the effect of doses of parathormone such as these to be continued for longer than fifteen hours. There undoubtedly is a fluctuation of the serum calcium level within the normal limits in those who have normal calcium metabolism.

EFFECT OF ADMINISTRATION OF PARATHORMONE OVER A LONG PERIOD TO PATIENTS WITH CATARACT.

Case No. 1.—F. O., a white male, 75 years of age, was first seen in February, 1927. There were incipient senile cortical cataracts present. The changes in the lenses consisted in water-split sutures with a few morgagnian globules in the clefts and faint lamellar separation in the inferionasal quadrants. The vision O. D. was 20/200 with +2.50 Sph., and O. S. 10/200 with +2.50 Sph. This great reduction in vision was due to a large area of macular degeneration in each eye. The condition was vascular in origin, there having been leakage from the capillaries in the choroid in the macular area. This case was selected because the psychic factor and the matter of visual acuity would not be important in judging the effect on the lens opacity. On October 30, 1928, when he was started on parathormone, the only changes from the previous year were in the graying of two of the morgagnian globules. Blood serum calcium was 11.0 mgms. per 100 cc. Parathormone was administered subcutaneously 1 cc. at a time, each dose representing 20 units, on the following dates. 1928: October 30. November 1, 3, 7, 9, 12, 14, 19, 21, 23, 26, 28, 30. December 3, 5, 7, 10, 12, 14, 17, 19, 21, 24, 26, 28. 1929: January 4, 7, 9, 11, 21, 23, 28, 30. February 4, 6, 8, 11, 13, 19, 25. March 4, 6, 8, 11, 18, 25. April 1, 9, 17, 24. Approximately 1,000 units of parathormone were used over a period of six months.

In April, 1929, there appeared fresh hemorrhages in the macula of each eye. It is believed that the parathormone did not con-

tribute to the production of the hemorrhages, but it was considered interesting at the time that the long continued use of the parathormone had not served to prevent their occurrence.

The water-split sutures and globules and lamellar separation remained unchanged during and after the injections. Reexamination was made on July 10, 1930, and there was observed extension of the split sutures in each lens, greater number of globules, with more that had become grayish and a moderate increase in the lamellar separation with haziness of the cortex in that area. The vision had decreased somewhat in each eye due to extension of the macular lesion.

Case No. 2.—J. M., age 62 years, male, white. Senile cortical cataract superimposed on heavily sclerosed nuclear opacity with amber reflex. Vision, O. S. with +2.00 Sph. 20/40; O. S. +1.50 Sph. 20/50. Blood serum calcium 9.4 mgms. per 100 cc.

September 4, 1928. V., O. D. 20/70	Acc O. D. 2.	20/40
O. S. 20/70	O. S. 1.50	20/50

In good general physical condition. Given parathormone 1 cc. (20 units) on following dates: 1928—October 30. November 1, 3, 7, 9, 12, 14, 19, 21, 23, 26, 28, 30. December 3, 5, 7, 10, 12, 14, 17, 19, 21, 24, 26, 28. 1929—January 4, 7, 9, 11, 14, 18, 21, 23, 25, 28, 30. February 1, 4, 8, 11, 13, 15, 18, 20, 25, 27. March 1, 4, 8, 11, 13, 15, 18, 20, 22, 25, 27, 29. April 1, 3, 10, 20, 22, 24, 29. May 6, 13, 20, 27. A total of 1,380 units of parathormone. Gradual progress of the opacity was noted. It became necessary to extract the cataract from the left eye in September, 1929, and from the right eye in March, 1930. Excellent visual result was obtained in each eye following operation. No retinal hemorrhages were evident.

In this case there was noted a steady progress of the lens opacity until useful vision was lost. The injections of parathormone apparently had no effect in absorbing or retarding the cataract. The cataracts were removed by operation, and the resulting vision with correcting lenses was excellent.

Case No. 3.—J. F., white male, 70 years of age, was seen first in February, 1927. At that time had in the right eye one water-split suture with two minute globules. The left eye showed no evidence of hydration of the cortex, only sclerosed nucleus. The vision was, with correcting lens, O. D. + 2.50 + .75 × 180°, 20/30; O. S. + 2.00 + .50 × 180°, 20/30. Physically he had some evidence of arteriosclerosis and myocardial insufficiency.

In February, 1928, there was observed the development of faint lamellar separation in the inferionasal quadrant of each eye with one minute vacuole beneath the capsule of the left lens. The water-split suture had remained practically unchanged. His blood serum calcium was 8.44 mgms. per 100 cc. He was given parathormone in 1 cc. doses (20 units) on the following dates: 1928—November 13, 15, 20, 27, 30. December 4, 6, 10, 12, 18, 20, 26. 1930—January 8, 10, 12, 15, 25, 28, 30. February 4, 6, 8, 11, 15, 20. March 4, 11, 27. May 3, 24. He was not as regular in attendance as the two other patients and pleaded the severity of the winter weather. He received a total of approximately 600 units of parathormone in six months' time.

Practically no change was perceptible during the six months while he was receiving the injections. The refraction and vision did not change. He was reexamined July 10, 1930, and it was found that the clear fluid of the water-split suture had been replaced by opaque material in a spoke which was smaller than the water-split suture. There was more lamellar separation. The vacuole remained unchanged. The vision of the right eye was the same, while that of the left eye had diminished to 20/50, not through progress in the cataract but through the development of a hemorrhagic retinitis.

COMMENTS ON THE USE OF PARATHYROID EXTRACT IN THE TREATMENT OF SENILE CATARACT.

There is no indication that the cataract which complicates tetany is analogous in any way to the average senile cataract. There is no evidence of parathyroid or calcium deficiency in these patients with senile cataract. The serum calcium is sufficient and undoubtedly furnishes enough calcium by diffusion to the aqueous, vitreous and lens. It is, therefore, unnecessary to attempt to raise the blood serum, aqueous, vitreous or lens calcium content. The use of parathyroid extract for this purpose is therefore not indicated in these cases.

Adams[2] felt there was a slight calcium excess in her cases of cataract but not of sufficient degree to cause the lens opacities. In our own series of cases the averages were within the upper limits of normal levels. It is hardly necessary to give these patients parathyroid extract in order to excrete the excess calcium, as this would not reduce the serum calcium level. The latter is raised by the hormone as increased excretion goes on.

It is not likely that parathyroid extract will accomplish the removal of excess calcium from the lens once opacities have formed. The hormone mobilizes calcium, but the great storehouse of calcium in the fine spicules of bone ramifying in the bone marrow will be depleted before deposits such as are found in cataract will be attacked.

It may be said that the series of cases on which parathyroid extract was tried is small, but in view of the facts that there is no need for the hormone and in the greater majority of cases was definitely contraindicated, there was no need for carrying the clinical test any further.

The evidence that a cataract has remained stationary for a certain period of time is not sufficient proof that the treatment has been effective in causing the standstill, as it is common knowledge that numerous cases of cataract do this without any treatment. For a certain treatment to be judged efficacious, it should in some cases cause absorption of existing opacities. Cataracts do not progress evenly and regularly, but go by fits and starts, there often being long periods of intermission between the stages.

The failure of parathormone to cause absorption of the lens opacities in these cases in no way reflects upon the value or efficacy of this agent in cases of calcium deficiency where it is indicated. The three patients were given sufficient units to get a physiologic effect when one considers that there was no evidence that they were not secreting a normal amount of the hormone themselves. Collip estimates this to be about 45 units daily. Aub has found that a refractory state is developed after a few weeks of treatment in many of his patients. In the average case repeated injections of parathormone cause a rise in serum calcium which is sustained for varying periods. Gradually there is less and less response. If the dose is increased there will again be a rise, but finally there is no further response. However, if the patient is then given NH_4Cl the serum calcium will again be increased, showing that the patient still has mobilizable calcium but has become refractory to parathormone. In the case of the three patients with cataract who were given parathormone for a period of six months there was no reason for continuing the injections any longer.

SUMMARY AND CONCLUSIONS.

The study of the relation of calcium to cataract was undertaken to clarify the situation in regard to this subject. The author

desired: (1) To assemble the latest information regarding calcium metabolism in general; (2) to know the importance of calcium in the normal growth and metabolism of the crystalline lens; (3) to learn the cause and prevention of tetany cataract both in infants and adults; (4) to find the reason for the deposition of calcium in senile cataract; (5) to discover whether patients with senile cataract suffered from calcium deficiency or excess; (6) to know whether calcium or parathyroid extract administration was indicated in cases of senile cataract, and finally whether their use caused clinical improvement.

The calcium in the aqueous represents the ionized and non-ionized diffusible fraction from the total calcium of the blood plasma. There is an extremely small amount present normally in the crystalline lens, but the importance of the element in combination with the other elements is out of all proportion to the chemicals involved. Calcium is greatly increased in the lens in senile cataract.

Calcium deficiency in cases of infantile tetany is undoubtedly responsible for the development of cataract in these cases. Even though the factors which produce and cure rickets are the same as for tetany, there is some difference that has not as yet been discovered between rickets and tetany, and cataract is linked up with the tetany rather than with the rickets complex.

No cases of tetany cataract have been seen at Babies' Hospital in the past two years. The widespread knowledge and use of the factors preventing and controlling rickets and tetany have led to a decrease in the number of tetany cataracts.

Three cases of postoperative tetany with cataracts from the thirty-eight to be found in the literature are reviewed. The important points to be learned are: (1) Prevention of removal of parathyroid tissue at the time of thyroidectomy. (2) Early treatment of such cases as show parathyroid deficiency. A great many of the latter are undoubtedly only temporary, due to vascular disturbances.

The pathogenesis of tetany cataract implies a reduction in the serum, aqueous and lens calcium content with electrochemical changes and disturbance of equilibrium of the inorganic elements in the lens causing changes in the labile colloidal solution of the lens protein. Once these changes are established, the administration of calcium or parathormone does not cause a restoration of the normal state of the protein. Not all tetany cataracts go on to

complete opacity. The prognosis in complete tetany cataract depends upon the operative removal of the cataract.

Coagulation of blood depends on the presence of ionized calcium. The coagulation and bleeding time of a series of patients with cataract was found normal.

There is no evidence of vitamin A or D deficiency in cases of senile cataract. The widespread use of vitamin D in various forms, and particularly viosterol, will help prevent and cure tetany but there is no indication for their use per se in senile cataract cases.

No cases of cataract in hypercalcemia have been found. A case of osteoporosis is cited in which there was excessive excretion of calcium but the crystalline lenses remained perfectly clear.

No evidence of calcium deficiency was found in the mother or child in a case of mongolism with cataracts.

In cases of senile cataract, calcium deposition most probably results from: (1) Very slow lens tissue death, dependent on nutritional deficiency; (2) degeneration of the lens protein with lipoid byproducts; (3) local concentration of calcium by adsorption to the large molecules of the lipoids, and (4) deposition of this calcium by change in the p H toward the alkalin side.

There was no evidence of tetany or latent tetany in a series of senile cataract cases.

Two separate series of cases of senile cataract were investigated as to their serum calcium levels. The first series of 28 males, aged 52 to 80 years, had an average level of 11.3 mgms. per 100 cc., while 25 females, aged 52 to 82 years, had an average level of 10.9 mgms. per 100 cc. The possible relation of the tendency to hypercalcemia in a certain number of these patients and the production of the cataract is interesting in view of Burge's work with ultraviolet light precipitating lens protein in the presence of an excess of electrolytes, particularly calcium. However, the conclusion of Burge[4] and of Adams[2] was that the concentrations of calcium necessary are not met with "in vivo."

A control series of normals and of those with other eye diseases than cataract had an average serum calcium level of 10.6 mgms. per 100 cc.

The second senile cataract series included 33 males, from 45 to 82 years. The average serum calcium level was 10.29 mgms., while for 50 females, ages 50 to 82 years, the level was 10.34

mgms. No variation could be found according to age, sex, type of cataract, visual quotient or complicating eye or general disease.

There is, therefore, no evidence of calcium deficiency as far as the blood serum values are concerned in cases of senile cataract.

The spinal fluid calcium in a small series of cases was found to be in proportion to the serum calcium of the particular cases. If the situation is analogous, one can say that there is sufficient calcium in the aqueous humor of these patients.

In the study of the endogenous calcium metabolism of a small series of cases of senile cataract, the relation of the intake of calcium in the diet was carefully calculated and compared with the output of calcium in the urine and feces. The results showed that they were in perfect or slightly positive balance when on neutral or slightly alkalin diet, but when on acid residue diet or receiving parathyroid extract injections they were in negative balance, excreting more calcium than they ingested. In other words, these patients were responding normally to the factors influencing calcium metabolism and were utilizing their calcium properly.

There was no indication for the administration of calcium salts to any of these patients. If patients with senile cataract are found to have normal serum calcium but are in negative calcium balance, it is better to correct the factor that is producing the latter than to administer calcium salts to them to make up the deficiency.

There was no reason for the administration of parathyroid extract to these patients, as they had sufficient serum calcium. Parathormone produced excess urinary excretion of calcium, but the excess calcium that was mobilized from the tissues undoubtedly came from the normal storehouse, the bony trabeculæ, where it is easily available, and not from the deposits in the cataracts.

Parathyroid extract in the form of parathormone was administered to three different series of cases of senile cataract over varying periods. No evidence of absorption of opacities or improvement in vision was observed.

These conclusions apply only to the cases investigated.

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DISCUSSION.

DR. A. D. RUEDEMANN, Cleveland, Ohio: I wish to mention our series of 136 cases of postoperative tetany in which there were eight cases of postoperative cataract. These tetany cataracts have a definite appearance in that there is granular distribution beneath the posterior capsule. In all cases they were females, two over 50, one 40, and the rest between 30 and 40 years of age. The first symptom the patients complained of was diminution of vision, which lasted anywhere from fifteen minutes to three or four hours, and during this period they had a marked blurring, during which they were unable to make out any object, and some complained of almost complete blindness. We feel this is a severe ciliary spasm, because putting the patients in a dark room and covering their eyes produced relaxation quicker than anything else. The tetanic type of cataract developed rapidly

in four cases, and all went on to complete cataract in a period of one month. Six have been removed surgically, five successfully and one having a prolapse of the iris postoperatively. These cases must be held under calcium control or the healing from surgery will be much prolonged. All cases had a very low calcium metabolism, the content running from 5.5 to 8.7, and there was a very low phosphorus content, which may play some part in the tetanic convulsions that these cases have. All the cases of tetany are controlled either by calcium or parathormone, or both, in conjunction with some other substance added to their diet, because some patients do not take up calcium lactate under ordinary conditions.

I believe Dr. Kirby is absolutely on the right track, that in many of these cases of senile cataract the progress can be stopped, that many of these cases, after they approach senescence, have a definite disturbance of the endocrine system, or disturbance of the metabolism of the body. There certainly is a disturbance of the metabolism one way or the other that brings on this type of catracts seen in postoperative tetany.

Dr. Frederick A. Davis, Madison, Wis.: I have been much interested in Dr. Kirby's excellent paper.

I have had the opportunity of observing a young married woman, age 33 years, who developed bilateral cataracts following a thyroidectomy, in whom the blood calcium was reduced to 4.9 mgs. per 100 cc. The patient was admitted to the University of Wisconsin General Hospital October 20, 1929, to the medical service of Dr. Sevringhaus, to whom I am indebted for much of the medical data contained in this report. The patient's chief complaint upon admission was blindness and a history of previous convulsions. Further history revealed that she had had a thyroidectomy in the fall of 1926. One month after the operation she began having convulsions, and about three months after the operation her vision began to fail. She reported having 26 convulsions in all over a period of two years, but she had had none during the two years immediately prior to admission. She became unconscious during an attack, the eyes jerked from side to side, she ground her teeth together, there were violent muscular contractions, and finally she fell into a deep sleep. Other symptoms were amnesia, changes in mentality, frontal headaches, deafness, marked gain in weight, and pruritis of the face, head and back, prickly feelings over the entire body, and stiffness of the muscles and joints.

The patient was a well nourished and rather obese female. There were no outstanding physical findings except a slightly increased growth of hair on the face and body, with dryness of the skin and hair. Chvostek's sign was positive in the orbital branch only; Trousseau's sign was also positive. The teeth and tonsils had been removed. The urine showed a faint trace of albumin; blood count was essentially normal. Blood serology: Questionable negative Wassermann on October 21, 1929, and 2 plus on October 28, 1929. Blood chemistry showed normal sugar and normal nonprotein nitrogen. Calcium content of the blood was definitely reduced, being 4.9 and 5.16 mgs. per 100 cc. on two determinations. Basal metabolism was plus 16 and plus 15 on two determinations. A diagnosis of tetany

from parathyroid deficiency was made by the medical department and the patient referred to the eye clinic for consultation.

I examined the patient at this time and found bilateral cataracts which had the appearance of soft, pre-senile type. The lenses were diffusely cloudy, giving the appearance of a homogeneous involvement of the entire cortex. They were almost mature at this examination, but since the anterior chambers were slightly shallow operation was deferred until the lenses appeared fully ripe. We also wished to observe the influence of intensive calcium treatment before operation. The eyes otherwise appeared normal. Vision O. D., 3/200; O. S., light perception, good projection. No view of fundus possible in either eye.

Patient was placed on calcium lactate, gr. x t. i. d., before meals, and a diet containing a maximum amount of cooked milk. Several readings of blood calcium following the administration of calcium lactate by mouth, as well as parathormone and viosterol, showed an elevation from 4.9 mgs. to 8.7 mgs. per 100 cc. Calcium gluconate was also used, but it did not appear so effective as calcium lactate. The patient refused to remain in the hospital longer than two weeks, and was sent home with the advice that she use calcium lactate, gr. xx, t. i. d. She stated she felt much improved generally by the treatment in the hospital; she was much happier and complained of less muscular discomfort.

She was readmitted to the medical ward one month after discharge, at which time she stated she was much improved. (Nervousness was less marked, twitchings less frequent, and the paresthesia of the skin had disappeared.) Blood calcium on readmission was 6.75 mgm., blood coagulation 3 minutes, blood pressure 60/100; Trousseau's sign was present, but Chvostek's sign had disappeared. Response to cathodal closing current was 2 m. amp. Examination of the eye showed slight changes; the cataracts appeared somewhat more mature; light projection was excellent in each eye, but the visual acuity had definitely diminished in the right eye.

A preliminary iridectomy was advised and successfully performed by a member of the house staff. There was no bleeding at operation. On the second postoperative day a hemorrhage appeared in the anterior chamber, which was followed by a destructive inflammation of the anterior segment of the globe, which finally quieted down. The vision was reduced to light perception only. Since the clotting and bleeding time in this patient was normal, it seems unlikely that the hemorrhage was due to lowered coagulability of blood. About twelve days postoperatively the patient developed a severe pharyngitis with a peritonsillar abscess, which probably came from a tonsil tag. The organisms responsible for the peritonsillar infection were undoubtedly present in the tonsil tag or nasopharynx at the time of operation, and this fact, together with the possible effect of the questionable Wassermann, beclouds the picture.

I do not think one can definitely draw the conclusion that the lowered blood calcium *per se* is responsible for the clouding of the lens, as Dr. Kirby has shown that there is no deficiency in blood calcium in patients with senile cataract. The repeated trauma to the lens which possibly ac-

companies the violent muscular contractions of tetany may be the etiologic factor, as suggested by Roemer.

Dr. C. S. O'Brien, Iowa City, Iowa: I wish first to congratulate Dr. Kirby on this very excellent piece of work, which has evidently been thorough and is proving of great interest to us.

I just want to say a few words on the relation of calcium to cataract. In normal lenses there has been a number of findings published. Burgin and Schlonika found a trace of calcium in ox eye lenses, Burgin finding 0.08 per cent in pig lenses, but none in the human eye. Dr. Salit found no calcium in normal ox and rabbit lenses. Adams found 18.5 mgms. in normal lenses. In cataractous lenses Dr. Salit examined 45 lenses, finding 35.4 mgms. of calcium per 100 per cent in these lenses, ranging from 2.8 to 100 per cent. He found—and this appeals to me as having a somewhat important bearing—no calcium in five cases of incipient cataract, and no calcium in one case of black cataract. Dr. Salit, working on the vitreous, found it contained 7.2 mgms. per 100 per cent. This was found in the eyes of an ox. The human vitreous, I think, has not been examined, or at least publication has not been made.

We found in Iowa that there was a decrease in the hydrogen ion concentration. It was found that the vitreous in senility became more alkaline as the individual aged. Also it has been found (this was done in Chicago in Dr. Brown's department at the Cook County Hospital) that the blood CO_2 content was also decreased with age. It may be that the theory of calcium formation or deposition in the lens in senile cataract may be more or less the same as A. G. Parsons' theory of the deposition of calcium in the bone, that the diffusible calcium of the blood exists as ions of dicalcium phosphate or calcium bicarbonate. When these ions arrive in bone where CO_2 content is lower than in the blood, calcium is precipitated as tricalcium phosphate.

It is my belief that calcium occurs in cataract as a result and not as a cause of cataract.

SYMPOSIUM ON TRACHOMA.

THE SPREAD OF TRACHOMA AND THE CAMPAIGN AGAINST IT.

Dr. Emile de Grósz,

PROFESSOR OF OPHTHALMOLOGY AT THE ROYAL HUNGARIAN UNIVERSITY OF BUDAPEST.

Trachoma is one of the most widespread diseases. It is of a chronic character and difficult to combat. It impairs sight and sometimes totally destroys it. It hinders children's learning, weakens the power of working and hence attacks the vitality of the nation. States in which the disease occurs sporadically have carefully to guard against the insidiousness of the disease, whilst countries where trachoma is endemic have to exert a vigorous energy against it. To combat trachoma is a national duty, but at the same time it is also of an international interest. During the fifteen years I was Government Commissioner in the fight against trachoma I repeatedly urged the launching of such an action. The Comité d'Hygiene de la Société des Nations (1925-26) dealt with the question of trachoma upon the proposal of Lutrario, but its discussions were relegated to the Office Internationale d'Hygiene upon the motion of Sir George Buchanan. The detailed accounts collected by the Section d'Hygiene were elaborated by Lutrario and Jitta and the Société des Nations published them.

One of the main themes of the XIIIth International Ophthalmological Congress was directed against the spread, and for the extirpation of trachoma, likewise this Congress witnessed the organization of the international anti-trachoma campaign. The organization was definitely formed in Geneva on the 26th of July, 1930, the honor of president being conferred upon me, that of the chief secretary on Dr. Wibaut (Amsterdam), and Dr. Tewfik (Cairo) has been appointed secretary. In the list of vice presidents, the United States is represented by Dr. Park Lewis, who at the Congress acted as delegate of the United States.

At this meeting Dr. P. K. Olitsky, representing the Rockefeller Institute, read a very interesting and comprehensive paper on the "Bacillus Granulosis."

The fight against trachoma has reached a new stage. A whole series of particulars is at our disposal. A new and vital organization has been formed, the aim of which is to lead and guide the campaign against trachoma. The Council of the Société des Nations appealed to the various states, asking their support for the organization. The Hungarian Government offered a prize of 2,000 Swiss francs for the best research work on the etiology of trachoma. The Rockefeller Institute and the Oscar Johnson Institute support scientific investigations, which constitute the basis of all advancement, brought about by great sacrifices.

The prospects are propitious; the task to be solved, however, a very hard one. In Hungary trachoma is responsible for 4 per cent of blindness. In Japan this percentage rises to 15. In Russia there are one million trachoma patients. In Poland there are about 400,000 trachoma patients. In Italy their number approaches 100,000, whilst in Egypt 90 per cent of the natives suffer from trachoma, and there are extensive trachoma nests also in the United States, in Tennessee, Kentucky, Arkansas and Missouri. But as it occurs elsewhere, too, trachoma constitutes a major public health problem.

Blindness in one of the United States has been the result of trachoma in 20 per cent of the cases. This entailed an annual outlay of $191,000 for, according to the law passed in 1922, every blind person is entitled to $300 yearly from the State.

The actions which were initiated for the protection of the population against trachoma have already shown some encouraging results.

As president of the International Organization for Combating Trachoma, this is the first time I have had the pleasure to make public statements, so may I be permitted to point out which are those great problems which await discussion and debate? The most important are the following:

1. Sometimes the establishing of a diagnosis is connected with difficulties, even in the present day.

After a lengthy and abundant experience, I confess to being a dualist; however, I separate follicular conjunctivitis from real trachoma. Differential diagnosis is, in the majority of cases, successful, as the infiltration of the conjunctiva, the deep situation of the follicles, eventual presence of a pannus, and complications at the rim of the eyelids confirm the diagnosis of trachoma, while the normal state of the frame of the conjunctiva, the superficial

appearance of the follicles, their displacement into a string of pearls, and the lack of complications suggest the diagnosis of follicular conjunctivitis. On the other hand, there are instances when follicular conjunctivitis is associated with some catarrhal process, rendering the formation of an accurate diagnosis impossible. In such cases we have to acknowledge the dubiousness of diagnosis utterly, and are compelled to defer the establishment of an accurate diagnosis for one or two weeks. The course of the disease decides the question inasmuch as with the approach of the total healing of the catarrhal process the basal affection becomes evident.

2. The second question is the establishment of the causative agent. Today this problem stands in the foreground, as the solution of it renders the diagnosis certain and the treatment successful. At least this is what we can hope for. Statements on this question will be made today by a competent author. For my part, I can state so much, that I have no doubt whatever as to the infectious character of the disease, and I strongly recommend the thorough and most scrutinizing study of the chlamydozoa, discovered by Halberstaedter and Provazek, the bacillus granulosis of Noguchi and Herzog's gonococcus theory.

Of course by this I don't wish to deny the importance of constitution. But I see recently there are some exaggerations met within this field, as the rôle of adenoidism is overestimated. Trachoma may affect even the healthiest men. We had ample opportunity of seeing such cases during the war, but we must admit that the course of the disease and the occurrence of the corneal complications are undoubtedly influenced by constitution.

There is a very slight progress in the therapy of this disease. Against the copious discharge of the eyes our most powerful drug today is nitrate of silver. Against papillary hypertrophy nothing surpasses in efficaciousness cauterizing with a copper sulphate pencil. The action of these drugs is efficaciously carried out by mechanical treatment: massage with glass tubes and particularly the expression of the granules by way of the Kuhnt method.

By this time we are already informed about the spread of the disease. The investigation of the Health Committee of the League of Nations, the reports heard at the Congress in Amsterdam and the publications given in the Revue Internationale du Trachome give a true picture of the vast spread of the disease.

Lutrario and Jitta elaborated the official data supplied by the health boards of the individual countries. From these particulars we became aware that not one single country could boast of being exempt from the ravages of trachoma, and there was not one single region which could be looked upon as immune.

The spread of the disease, however, is greatly influenced by the culture and economic state of the population. Trachoma is a disease brought about by poverty and want of culture. The particulars collected by the League of Nations concern trachoma patients who are registered as such, but their number does not nearly approach the number of those actually suffering from trachoma. For instance, there were 67 trachoma patients reported in France in 1926, but at the same time in one single clinic, that of Aubaret in Marseilles, there were 387 trachoma patients under treatment.

The official data are the nearest to reality in Lithuania and Japan. The whole population in Lithuania was subjected to medical examination for trachoma, and it was found that 34 per cent of the inhabitants were suffering from it. In Japan ten million people have been examined, from the whole population, and 700,000 trachoma patients were recorded.

Such general eye tests were performed in Hungary in 1883, when 100,000 of the inhabitants of three districts of a county were tested, and the same was done in 1928 in two districts of the county of Zala. This method, however, entailed great expenditure, it took a long time and in most countries there is a scarcity of money and a lack of sufficiently competent doctors.

It is not to be doubted, however, that absolutely reliable data may be gained in such a way only.

Those to whom the matter was referred by the XIIIth International Ophthalmological Congress availed themselves of other methods too and derived full particulars from the most diverse sources. These sources were as follows: The testing of the eyes of school children, recruits, laborers, emigrants, likewise patients calling for free outpatient treatment at the clinics. Of course, this method enabled one to get thorough information as to the spread of the disease, for if there was no trachoma among school children and recruits then it was fairly probable that it could not be very common among the population either. Where there are about 10 to 15 per cent of trachoma patients among general eye patients it is highly probable that there are a good many trachoma patients

also among the population in general. These are the methods which are prescribed by the Hungarian law, accepted by the parliament of 1886 and which I—being then Government Commissioner—decreed to be used widely in an enactment issued in 1903.

Wibaut, availing himself of all these particulars, has drawn a map which displays the geographical distribution of trachoma. As the particulars obtained from the single countries are very varying, this has been only an experiment, which gave no true conception. I think I will bring forward a few examples taken from such states, from which there are fairly reliable data at hand.

In Italy the recruits and the military in general were subjected to scrupulous examination, which elicited that the average rate of morbidity, fixed as .88 per cent, rose to 3 to 6 per cent in Sardinia and Sicily, while in Upper Italy it is considered less. Maggiore roughly estimates the number of trachoma patients at 80,000. In Poland there are about 400,000 trachoma patients. Out of 885,476 school children, 14,463 have been found to suffer from trachoma; in the orphan homes the rate of the trachomatous children rose to 15 per cent.

In Russia, out of the total population amounting to 147,000,000, 1,000,000 trachoma patients exist; in some districts 50 per cent of the population are ill.

In Slovensko, which at present belongs to Czechoslovakia, out of 3,000,000 inhabitants, 60,000 persons have been found suffering from trachoma. As I already mentioned, the most reliable data have been supplied by Japan; the morbidity rate of the general population, of the school children, of some employment branches and of certain stages of life are separately revealed in graphic reports.

Very interesting particulars on trachoma were published in Public Health Reports (No. 8, 1928), by Paul D. Mossman and also by Dr. William Wilder in the May number (1930) of the American Journal of Ophthalmology.

Naturally I know best the conditions relating to the spread of trachoma in Hungary, where reports of cases and eye examinations have been made since 1883, and where I acted for fifteen years as Government Commissioner. The eye examinations made, on the ground of the enactments issued in the year 1928, brought to light that in mutilated Hungary—as is generally known, Hungary has lost two-thirds of her whole territory

through the force of the Treaty of Trianon—altogether 12,000 trachoma patients were notified, against a whole population of 8,500,000. Out of 829,284 school children, 3,257 have been registered as suffering from trachoma. These data, however, do not come near to the actual conditions. In the county of Zala there are two districts, in which some eye specialists brought to light that, according to the registrations and reports, 10 per cent of the population suffered from trachoma, while in reality 30 to 40 per cent have been found. This should serve as an example from which we ought to deduce the object lesson that in collecting data we have to avail ourselves of all possible sources.

In the university eye clinics trachoma patients were found among the outpatients at the following rate: In Budapest, 2 per cent; in the eye clinic of the Szeged University, 10 per cent (the environment of Szeged is the chief bed of trachoma); in Debrecen and Pécs (both cities are seats of universities), 5 per cent.

Of course, the collection of data is reasonable only where it is immediately followed by the application of adequate prophylactic measures.

In the bringing in of laws, Hungary took the lead; in the execution of the laws and enactments, Prussia and Japan are showing the way. Very edifying is the Egyptian system, and much can be learned from the action of the United States.

The first separate trachoma law was passed by the Hungarian legislature in 1886 (Coloman Tisza was Prime Minister at that time). The provisions of this law hold good, even at the present day. They include the following important points:

1. The nursing expenses of all indigent trachoma patients, treated either in a hospital or at home by order of the authorities, are borne by the State treasury. 2. In areas where the complaint is found to a more considerable extent, ophthalmologists will be sent out with the duty of ascertaining the extent of the disease, to organize treatment in the communes and in the meantime to treat the serious cases, as well as to instruct in the therapy of the disease such district and parish doctors as are not sufficiently versed in the treatment of this complaint. 3. Temporary eye hospitals will be erected for the period required. 4. With a view to the training of communal doctors, parish doctors and medical officers in the pathology and therapy of trachoma, the Minister of the Interior is empowered to arrange courses of instruction for which material will be distributed to the partici-

pating physicians and remuneration fixed for the communal and parish doctors. 5. The costs of all these measures will be defrayed by the State treasury.

As Government Commissioner, I endeavored to increase the number of eye hospitals. Some of them have remained on the ceded areas. We have had to provide accommodation for eye patients in mutilated Hungary.

In Budapest there are at present 593 beds for the accommodation of eye patients, of which 131 are for trachoma cases. In the provinces there are 583 beds, of which 135 are allotted to trachoma patients.

Prussia—In Prussia defensive measures against trachoma were first taken in 1889 on the basis of personal negotiations between the representatives of science and the ministry, the main lines of the scheme being drawn up by Professor Kuhnt of Königsberg, the chief points of which are as follows:

1. Extension courses for the doctors. 2. Foundation of trachoma wards. 3. Eye examination in schools. 4. After treatment of the patients discharged from the eye wards. 5. Instruction of the population. 6. Gratuitous treatment of trachoma patients. In the past few years forty courses have been held, in which 700 doctors have been instructed. The sum of 350,000 reichsmarks annually has been allotted to meet the expenses of the campaign. The task of the competent circles is the organization thereof, the control and management being the affair of the central authorities. The defensive campaign has a double task: to cure the patients and to improve hygienic conditions. Nurses have proved particularly valuable in connection with this treatment. The expenses of a nurse are fixed at 800 marks annually, to which sum the State contributes 200 to 250 marks while the remainder is covered by the communes and districts.

The family group is the chief breeding place of the trachoma scourge. At Masurem, in a family of eleven persons, ten were suffering from trachoma. All lived together in a single room, using a single rag for a towel. Of 4,159 children suffering from trachoma, 2,849—i. e., 68 per cent—came of families infected with trachoma. The poverty of these families, their ignorance, their unhealthy dwellings have all assisted the spread of the disease.

The result of the introduction of defensive action was quickly apparent. In three strongly infected districts of East Prussia

(Königsberg, Gumbinnen and Allenstein), the number of school children suffering from trachoma was considerably reduced. In the year 1898 the number of school children afflicted with trachoma was 40,047, of which 16,793 were serious cases. Five years later there were only 5,515, of which 1,301 were serious cases. Thus in five years the number of trachoma patients was reduced by 34,500.

The number of trachoma patients reported by the district doctors in the government district of Königsberg amounted to 1,060 in 1913, 520 in 1920 and 189 in 1926.

A similar improvement was manifest in the recruits. Whereas in the year 1890, on the occasion of the periodical levies, 1,438 trachoma patients were found among those liable to military service, in 1912 the number amounted to 212 only.

In like manner, the number of trachoma patients in the provinces of West Prussia has also diminished. In the year 1913, in the governmental district of Mariawerder, 2.7 per cent of all the school children suffered from trachoma, while in the year 1924 only 0.15 per cent were infected.

From 1914 to 1918 there was an increase in the number of trachoma patients, as the doctors and nurses were all required at the theater of war. At the conclusion of peace, however, the campaign was re-started, not without some success. Moreover, on the occasion of the discussion of the trachoma question in the Hygiene Committee of the League of Nations, it was shown that the spread of trachoma in Prussia has decreased by 90 per cent.

Italy—In Italy the defense against trachoma is generously supported by the State, which grants loans free of interest as well as subsidies to the suburbs of the capital and the provinces for the erection of trachoma consulting rooms, hospitals and schools. It provides for the further training of the doctors and the enlightenment of the people. In 1926 there were founded, in addition to the clinics, 208 trachoma consulting rooms and 28 hospital wards for trachoma cases. In one province a special institute has been established for the campaign against trachoma. Social insurance also cooperates very largely in the struggle.

Japan—Japan has had a trachoma law since 1919. This has rendered it possible to check the spread of the disease and to organize an extensive defense against the scourge. Of her 61,000,000 inhabitants, Japan examines about 10,000,000 annually

for trachoma, while she examines the entire population of the endemic areas (about 5,000,000), 2,500,000 school children, 300,000 recruits, 500,000 factory workers, as well as about 1,000,000 persons employed in such occupations as might contribute to the spreading of the disease. In certain provinces 30 per cent of the population suffers from trachoma, while in others 2 per cent only are affected. Of the recruits 12 per cent were suffering from trachoma and of the school children 14 per cent. Of the patients treated in the university ophthalmological clinics, public hospitals and eye wards, 24 per cent suffered from trachoma; 12 per cent of these were severe cases. The law of 1919 makes curative treatment compulsory. There are 1,268 institutes at disposal for this purpose. The sum of half a million yen annually is spent in the campaign against trachoma. The result is already perceptible, as the number of trachoma patients is decreasing and severe cases are becoming rarer as time goes on.

Egypt—In Egypt 90 to 95 per cent of the native population suffer from trachoma. The campaign, which was organized by MacCallan and financed by Sir Ernest Cromer, is being carried on with great energy. At the present time 29 permanent and 14 traveling eye hospitals with 849 beds are at work. About 381,790 outpatients and 12,967 inpatients annually are treated. Treatment has also been organized in 30 schools. The central laboratory in Cairo completes the system.

Holland—In the Netherlands trachoma existed only in Amsterdam, among the immigrant Jewish population. That the disease there was of a family character was also proved, for of the 4,685 members of the families of the trachoma patients 1,610 (i. e., 34 per cent) were suffering from trachoma. The children take the disease in infancy. Success has been achieved in checking the spread of the disease by means of hygienic regulations, as by providing ophthalmological consulting rooms, the employment of an eye doctor, the distribution of towels, and the improvement of the living conditions generally, the number of children suffering from trachoma in the Jewish schools has fallen from 35 per cent to 8 per cent during the past five years.

United States of America—In the United States defensive measures against trachoma were first instituted systematically in Kentucky in 1912. Later, upon the proposition of McMullen, hospitals were founded in Virginia, Tennessee, Arkansas and Missouri.

The trains carry about 50 itinerant consulting rooms, and these afford systematic enlightenment to the traveling population. Besides therapy, the hospitals and consulting rooms take part in prophylaxis, in accordance with my proposition made in 1903.

The eyes of immigrants are very strictly examined and those found suffering from trachoma are not allowed to land. From 1910 to 1924, of 15,000,000 would-be immigrants, 25,000 were rejected on account of trachoma.

In view of all these considerations, in the interest of the campaign against trachoma, I find the following measures necessary:

1. In order to ascertain the spread of trachoma the duty of reporting all cases is essential. Experience has taught us that the duty of reporting cases is neglected whenever no measures follow such steps.

Where the defense has a well considered and suitable foundation a so-called orientative (preliminary) eye examination is required, especially with regard to school children. Moreover, valuable data are furnished by examining the eyes of the relatives of school children, factory workers, returned emigrants, the inhabitants of tenement houses, and those in places where military conscription is in force on the occasion of the enlistments. In accordance with the data at our disposal, specially instructed ophthalmologists should be sent to the districts where trachoma is endemic who, by means of controlling examinations, should ascertain the state of affairs, and in case of necessity carry out a general eye examination of the whole population within smaller limits.

Precise instructions for the investigating physicians, for both the orientative and the controlling examinations should be elaborated, with regard to carrying out the investigation and what principles are to be considered in making the diagnoses.

2. Defense should be instituted without delay on the basis of the data furnished. Ambulatory treatment should be entrusted to the parish doctors after a suitable course of instruction of at least fourteen days' duration. Places where trachoma is prevalent on a larger scale must be reserved for trachoma treatment by experienced ophthalmologists.

Trachoma treatment is also given in eye hospitals, in eye departments possessing separate rooms for trachoma patients. The introduction of such establishments into the defense movement is of essential importance. The leaders of the eye clinics, eye

hospitals and departments, or their representatives, should from time to time visit infected areas to support the ophthalmologists in charge of the ambulatory treatment with their consultations, and arrange for the accommodation in hospitals of such persons as require hospital treatment and take their share in the preventive as well as in the ambulatory and hospital treatment. The hospitals are not only the most successful centers of treatment, but it is also their vocation to be intermediary stations in the work of defense.

3. The campaign against trachoma is directed from the center (Ministry of Welfare and Public Health). The trachoma inspector, appointed and authorized by the Government and competently supported, is at the head of this movement, particulars of which can be ascertained in the locality, where the defense is accommodated to the local public characteristics.

4. Persons of unsound mind suffering from trachoma should be relegated to separate trachoma departments of the asylums, care being taken that they are placed under the treatment of a specialist.

In districts largely contaminated with trachoma, special schools should be provided for children afflicted with the disease, while the trachoma patients of the orphanages and institutions should be treated in a similar manner.

To accommodate in hospitals those mentioned in the preceding paragraph is impractical both from the pedagogic and hygienic points of view, since the treatment of trachoma is a lengthy process and there is danger of its reappearance years afterwards. Besides this, so to accommodate the aforesaid is impracticable also because there is no suitable bed space at the disposal of the hospitals.

5. For instruction in the diagnosis and therapy of trachoma great importance is attached to courses of instruction devoted to further education at the universities, and special courses of instruction on the subject of trachoma should be arranged.

6. The defense against trachoma should be coordinated through international association. It is especially important that we should have a convention with each of the neighboring States.

7. Scientific research on the subject of trachoma should be promoted. With this object in view, the Royal Hungarian Minister of Public Welfare and Labor has instituted an international prize of 2,000 Swiss francs, to be awarded for the best independ-

ent treatise on the etiology of trachoma, the contents of which treatise manifest valuable progress in the above mentioned sphere.

The campaign against trachoma is long and fatiguing; success can only be achieved by patience and perseverance.

Prosperity, civilization, making diagnosis surer and treatment more successful, together with the improvement of hygienic conditions, the establishment of trachoma consulting rooms, trachoma schools, eye hospitals, and popular enlightenment, are the means whereby we may attain success.

The organization founded at Scheweningen in 1929 and extended to Geneva in 1930—i. e., the Anti-Trachoma Organization —will serve to promote efficient defense.

The aims of the organization are set forth in the statutes as follows:

The Leagues objects are: (a) To further the cooperation of the various organizations for the prevention of trachoma; (b) to work with the international public health organizations; (c) to follow all measures concerning the anti-trachoma campaign, to second these measures and to spread them; (d) to organize conferences, to establish the scientific bases of the anti-trachoma campaign, to elucidate problems of the etiology, diagnosis, treatment, extension and prevention of this disease, as well as the scientific and social questions therewith connected, finally to prepare the necessary legislative measures.

The Executive Committee of the organization is as follows:

President: Dr. Emil de Grósz, elected for the period of five years.

Secretary General: Dr. F. Wibaut (Amsterdam).

Secretary: Dr. M. Tewfik (Cairo).

Vice-Presidents: Professor Angelucci (Naples), Professor Birch-Hirschfeld (Königsberg), MacCallan, M. D. (London), Professor Marquez (chairman of the International Ophthalmological Congress at Madrid), Dr. Morax (Paris), Dr. Park Lewis (Buffalo), Professor Szymanski (Wilna), president of the Upper House of Poland.

Other members of the Executive Committee:

Dr. Brandes (Antwerp), Professor Kadlicky (Prague), Professor de Lapersonne (Paris), Professor Myashita (Tokio), Professor Van der Hoeve (Leiden).

The representatives of the Hygienic Committee of the League of Nations, Dr. Lutrario, former chief of the Italian Medical

Department, and Dr. Jitta, chairman of the Dutch Board of Health, will join the organization. The representative of the "Association Internationale de Prophylaxie de la Cécité" will be Professor de Lapersonne (Paris) and that of the International Ophthalmological Council, Professor Van der Hoeve (Leiden).

The Executive Committee will hold a session at Budapest in June, 1931. The entire organization will hold a general plenary session at Madrid in 1933. The United States with its great intellectual and material resources is in position to contribute enormously to the promotion of the work of the organization.

In the Rockefeller Institute research is being diligently prosecuted to discover the cause of trachoma, while the Oscar Johnson Institute in St. Louis a short time ago received a bequest of $250,000 for trachoma research. Every sign points to the United States as the ultimate solver of the trachoma problem, thereby conferring an invaluable blessing upon humanity and eminently contributing to the cause of progress and civilization.

SUMMARY OF OBSERVATIONS AND EXPERIENCES WITH TRACHOMA IN EASTERN KENTUCKY.

J. A. Stucky, M. D.,

Lexington, Ky.

In complying with the request to present a brief summary of my observations and experience with trachoma in Eastern Kentucky, it is necessary to call attention to the locality, the people and their living conditions twenty-five years ago, their general physical, as well as eye conditions, the work done in their cabin homes and in tents carried with us as well as along the roadsides and the measures instituted then for their relief.

For many years an increasing number of patients with eye diseases had been coming to my office in Lexington, from the eastern part of the State, and being impressed with their genuineness, simplicity, honesty and sincerity, as well as the fact that the majority of them could neither read nor write, I determined to go into this section and see for myself what the conditions were and what was causing the increasing number of eye conditions I was seeing.

I found a country where the only[1] viaducts were aqueducts—the creek beds—these having known no shaping by the hand of man, and when the creek bed became impassable you wound along the edge of the stream or over the steep mountains by a narrow trail. Among these mountains I found a people who had always been Americans and since the days of Daniel Boone had changed very little, not laborers or salaried folk, but soil owners, home makers, by no means what is known as the "poor white trash" of the South, but a race of brave men and women, remains of the Revolutionary type. Their homes, rude log cabins, with the loom, spinning wheel, lard kettle, candle mold and squirrel rifle, unchanged in a hundred years. The women wore sun bonnets, rode behind their lords and masters, on horse or muleback, and stood at the table waiting on the men while they ate. Strange old Elizabethan phrases of speech were their common language. One could not believe without seeing it that such a condition could exist in America, but having seen it one would understand

why they were uneducated. There were no schools because there were no roads; there were no roads because there were no taxes; there were no taxes because there was no money, and so it ran—these mountain people, pocketed in this section of the wilderness, much as Boone saw it and left it, had been sidetracked or set aside by civilization as we know it.

I found full-grown men of powerful body and keen mind, showing a breadth of justice and mental view simply astonishing, as their total book knowledge would not equal that of a five-year-old child in the average community as we know it. What did it mean? Simply arrested development; their little cabin homes, rude huts of one door and no windows, a lean-to chimney, sometimes in the more fortunate families a lean-to room built against the side of the cabin, serving for a kitchen, and in such huts as these lived families ranging in size from eight to twenty. In these one or two rooms they lived, ate, slept, cooked; the entire family often using the same towel for days at a time, drinking from the same cup, sleeping several in the same bed, or all lying side-by-side on pallets on the floor. Everything they ate was cooked in grease, a cooking stove was rarely seen, everything being cooked on the open fireplace and large quantities of sorghum molasses with hot soggy bread were consumed with large quantities of meats and vegetables cooked in grease, impairing their digestion, lowering their body resistance and making them an easy prey to any infectious disease.

Due to the hard labor they had to undergo in ekeing out a living from the steep land they must till, they aged rapidly, especially the women, on whom the harder work most often fell, and women of thirty were toothless, wrinkled and haggard, looking every day of fifty or sixty. There being very little pasture land in this section, few cows were kept, hence milk and butter were very scarce. Eggs and chickens were bartered at the little stores for cheap calico, thread and such household commodities as they could buy. If sugar or canned goods were obtained they were of the cheapest variety, which added to their already unfortunate circumstances rather than aided them.

Three elements which constantly presented themselves at the first clinics and kept us on the qui vive from start to finish were the elements of uncertainty, expectancy and danger, and while details cannot be given in this limited time, it must be said that the curiosity which led me to investigate this section was lib-

erally paid for in these three elements, and in the hard, rough, grand and many times perilous journeys.

In none of my clinics or trips in this section, forty-five miles from a railroad, did I meet with many cases that I could with confidence diagnose as acute trachomatous infection. As I found this disease, it was chronic, sometimes months and years intervening between the time of its incipiency and the terminal stage of cicatrization with one or several of the inevitable sequelæ.

In the beginning trachoma has the appearance of ordinary conjunctivitis.[2] If extensive hypertrophy has occurred, if the conjunctiva cannot be stretched and the blood vessels plainly seen, especially if other members of the family, or anyone in the neighborhood with whom the patient comes in contact shows evidence of conjunctival cicatrices, I at once convict the patient on circumstantial evidence and treat the case as trachoma. It is difficult to decide whether a case in the acute stage is trachoma, but if the conjunctiva is thickened and inflamed, and there are other cases of the disease in the family or neighborhood, the case is regarded as suspicious.

In the second stage, the differential diagnosis between trachoma and folliculosis of the conjunctiva is based on my observation that in simple follicular hypertrophy (that is, adenoid hypertrophy of the subepithelial layer of the conjunctiva) the conjunctiva is easily and evenly put on the stretch, the whole membrane being elastic and the blood vessels visibly outlined. This is not the case in true trachoma. This pathological differentiation is applicable in the study of every case. I prefer the term adenomatous conjunctiva, inasmuch as it covers the ground, whether or not there are inflammatory phenomena present, and does away with the necessity of distinguishing between folliculosis (without inflammation) and follicular conjunctivitis (folliculosis plus inflammation).

Hyperplasia of the conjunctiva is characteristic of trachoma, but not pathognomonic. It is this hyperplasia which causes numerous fine folds in the membrane, giving it the thickened, cracked, often tessellated appearance of many papillæ crowded together, whence the disease gets its name (granular, rough). Pannus is pathognomonic of trachoma, being largely if not entirely the result of irritation by the roughened lids.

In the first four years of my semi-annual clinics, as we journeyed hundreds of miles on horseback or in a springless mountain

wagon, stopping at the humble mountain cabins for water or directions as to the way to go, sometimes sleeping in these homes when overtaken by night, or when the sudden "rising" of the creeks or river made continuing the journey unsafe, I have seen numbers of cases of trachoma, as evidenced by the gummy appearance of the eyelids, or by ptosis of the upper eyelids, photophobia or profuse lacrimation, and the usual answer to my question, "What's the trouble with your eyes?" would be "Twarn't nothin' much, only jest sore eyes, which gits powerful weak at times." To my next question as to how long this condition had existed I would hear, "Oh, I've follered havin' sore eyes fer quite a spell; they gits mighty nigh well at times, and then they busts out wuss than ever." Such patients as this did not come to my clinics nor seek medical aid, for the pain was not great enough nor vision bad enough to prevent their making the "craps" or "lumberin'," and they *must* live. With no knowledge of farming it was hard for them to make a living, hence they were insufficiently clothed and fed, and did not have the means or facilities to get anywhere for the right kind of clothing, food or medical care. The pathos and tragedy of their state was that if they ever received help it would have to be carried to them, because they were mountain-locked, misunderstood, misrepresented, crowded back by a civilization that had never given them a fair chance or a square deal.

After battling with these conditions for four or five years, through my semiannual clinics conducted in the cabins, tents and by treatments given along the trails as we journeyed, I made an appeal (a real S. O. S.) at the meeting of the American Academy of Ophthalmology and Otolaryngology in Indianapolis in 1911 for advice and help, with the result that the A. M. A., the Kentucky State Board of Health and the U. S. B. P. H. came to our rescue and established five temporary hospitals in Eastern Kentucky, and within ten years the disease was practically arrested and hundreds saved from a life of blindness and dependence.

In 1912 conditions revealed by the U. S. Government reports showed that of over 4,000 examined in five counties, 12.5 per cent had trachoma.[3 4]

To my clinics only those with the most painful form of the disease would come, and in September, 1912, of the 374 cases examined, 113 had trachoma, 40 per cent had corneal ulcer, 25

per cent had photophobia, 10 per cent had entropion, 2½ per cent had symblepharon and 25 per cent had impaired vision ranging anywhere from slightly defective to total blindness, all of which were sequelæ of the disease. These patients ranged in age from small children in the mother's arms to old age.

Trachomatous patients coming into Ellis Island from the European ports were quarantined, but on a visit to this station, of the 114 cases I examined, not .5 per cent were comparable in severity to the average case I saw at my clinics in the mountains of Kentucky. Besides the ravages of trachoma, I saw many suppurative diseases of the ear, nose and throat with enlarged tonsils and adenoids, though probably more of the natives were afflicted with trachoma and hookworm and their sequelæ than any other two diseases.

The condition confronting the state and federal government was of more than intense scientific interest. It was at once a humane and economic one and could not be viewed either from a political, sectarian or sectional point of view.

In these early days, Dr. Herbert Harlan of Baltimore visited the government hospital at Hindman, helping and advising in the operative work, and later Dr. F. Park Lewis of Buffalo spent some time with us, both of these men rendering invaluable aid and giving encouragement to all connected with the work. Many other oculists were invited to come, but some of them felt that if the conditions I had described at Minneapolis, in 1913, existed in this section, the eastern part of Kentucky should be quarantined and all the workers with it.

We have seen in our clinical work that[5] "vitamin A" promotes tissue formation, increases the blood platelets, prevents infection, notably of the eye, sinuses, air passages and lungs, but a deficiency of vitamin A causes retardation of development, induces a susceptibility to diseases of eye and ears, causes excessive growth of lymphoid tissue and gives rise to dullness or perversion of special senses.

A deficiency of vitamin B impairs the appetite and digestion, produces constipation, causes subnormal temperature (cold hands and feet) and various other manifestations referable to the nervous system. A proper amount of vitamin B in the diet relieves these conditions; a sufficient quantity of vitamin D controls the calcium equilibrium and regulates mineral metabolism. A de-

ficiency of this vitamin in the diet causes muscular weakness and instability of the nervous system.

The combined deficiency of vitamins A, B and D, especially of A, has been noted in the larger per cent of the cases of trachoma treated by me.

Proper feeding after grattage has given most satisfactory results, in that there were both fewer and milder relapses and almost none of the usual destructive sequelæ. The treatment of trachoma is primarily and necessarily surgical and the immediate after-treatment is most important and can only be administered by trained assistants, and the patient should have hospital care for some days to prevent synechiæ and other complications.

Too much cannot be said of the splendid work done by the U. S. B. P. H., under the direction of Surgeon John McMullen, but I do not believe his surgical work, which consisted of grattage only, would have given the lasting benefit it did had it not been for the fact that the patients were kept in the hospitals for some days and had a balanced diet while they were being treated, thus building up their body resistance and teaching them how to live. For a great majority it was the first time they had ever had a practical demonstration of nutritional, sanitary and hygienic living, and during the time of their stay in these hospitals they learned much of value to them when they went back to their little cabins.

With the character of physical and mental Anglo-Saxon stock we had to deal with in the mountains of Kentucky, I stated in the beginning that I felt confident the disease could be arrested and eradicated by the methods used in the floating ophthalmic hospitals of Egypt, but in order to do this effectually we must have the combined efforts of the medical man, the trained nurse and the school teacher. After about fifteen years of active work we see today not one-tenth of the active cases of trachoma with which we had to deal in the beginning.

For the past three or four years, instead of the five temporary hospitals, the United States Government co-operating with the Kentucky State Board of Health has maintained only one trachoma hospital, with a capacity of thirty patients, at Richmond, Madison County, in the foothills of the mountains of Eastern Kentucky, and this, under the direction of Dr. Robert Sory, is able to care for the remaining active cases of trachoma seeking

treatment. Here, in addition to the necessary surgical and medical treatment, they have a balanced feeding and are taught by precept and example proper sanitary and hygienic living, having ample room for outdoor life in the fresh air and sunshine.

The average case of trachoma which has come to me in the last five years is an acute exacerbation of the chronic disease and is a pitiable picture, intense photophobia, lids swollen with well marked episcleritis and keratitis, often with marked pannus, the everted lids showing a mass of soft, acutely inflamed conjunctiva on top of a fibrous, papillary or hyperplastic condition with a vascular fungus appearance. The iris is usually thickened, presenting a muddy appearance and showing no reaction to light, a typical picture of conjunctivitis, keratitis and iritis. Such cases are treated by instilling a local anesthetic, usually 2 per cent butyn solution, in the eyes at intervals of three to five minutes, for three or four times; then the everted surface is grattaged and touched with a solution of trichloracetic acid on a cotton-covered applicator, the grattage continuing until the bleeding stops. The grattaged surface is then gently touched with a 5 per cent mercurochrome solution, applied in the same manner as the trichloracetic acid, and the eyes irrigated and ointment applied. If the pain following the treatment is not relieved by the local anesthetic the patient may be given an anodyne with instructions to bathe the eyes frequently with equal parts of saturated solution of acidi boracic and normal saline. I seldom use the lapis divinus pencil as I formerly did. For the iritis and intense photophobia, protein therapy, injections of milk either natural or artificial, have given gratifying results, 5 cc. being given an adult and repeated every second or third day until there is no reaction. Dietary treatment consists of a well balanced diet: whole wheat bread or corn bread of unbolted meal, green vegetables, fruits and milk, with at least one drachm of cod liver oil daily before meals. This treatment must be rigidly adhered to for from three to six months, and in patients who have followed this regime I have few recurrences of the disease.

G. H. Bell (Arch. Ophth., Feb., 1924) shows conclusively the influence of diet in many clinical cases of corneal ulcer, keratomalacia, glaucoma, choroiditis and other ocular diseases, and the same positive statement may be made of trachoma. The condition found in nearly all the cases I have seen is that of hypo-alkalinization and hypo-calcemia or acidosis, rapid and satisfactory

results following the remineralization and giving of food rich in minerals and in vitamins A, B and D. Clinical observations confirm the inherited tendency to the disease, and prenatal care, especially as to feeding, should be given the expectant mother who has trachoma.

I have never seen trachoma in its endemic, epidemic or pandemic form, and I have never seen a case in which there was unmistakable evidence that it was contracted by contact with one having trachoma. I believe genuine trachoma will eventually be proved to be a deficiency disease, a term sanctioned by custom and embracing disorders due to faulty or ill-balanced food.

I am still of the opinion that trachoma begins as an adenomatous conjunctival irritation, due to improper feeding and impaired metabolism, and I join with a united profession in the hope that we are near to isolating the specific etiological factor in its production.

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RECENT STUDIES ON BACTERIUM GRANULOSIS: ITS SIGNIFICANCE IN HUMAN TRACHOMA.*

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At the first meeting of the International Union Against Trachoma, held in Geneva last July, the writer presented a paper[1] in which the work of Noguchi together with its later elaboration by others was reviewed and the relation of *Bacterium granulosis* to human trachoma was discussed.

In this communication I shall summarize the views expressed at the Geneva meeting and include the results of later experiments, all of which relate to the subject to be discussed today. I owe thanks to your president, Dr. William H. Wilder, for the privilege and honor of taking part in the symposium of the Academy.

The earlier work of Noguchi is now too well known to require a detailed description. I wish to recall, however, his selection of five patients among American Indians who were afflicted with trachoma clinically and pathologically indistinguishable from the disease in the old world. The suspensions of conjunctival tissue of the patients yielded, after artificial cultivation, a number of microorganisms of which only one species, hitherto undescribed, was pathogenic for monkeys and apes, inducing in them a granular conjunctivitis closely resembling trachoma in man. Four of the five cases yielded this microorganism, called by Noguchi *Bacterium granulosis;* the fifth (No. 2),[2] from whom the microorganism was not obtained, was the one later reported by Lindner[3] as being free from lesions. At that time Lindner pronounced two available patients of the remaining four as having typical trachoma.

Noguchi described *Bacterium granulosis* as a motile, aerobic, Gram-negative, minute bacillus with a single polar flagellum and

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with distinctive fermentation reactions; growing best at 15 to 30 degrees C. on blood agar plates or in leptospira medium. The organisms formed small, semi-transparent, shiny and bluish-gray, moist colonies. Noguchi reported that after production, by means of cultures, of the experimental disease in one eye of each of several monkeys, characteristic infection followed in the other eye, and, in addition, that the microscopic changes in the conjunctivæ of the affected monkeys corresponded closely with those of human trachoma. The absence of pannus in the experimental disease was not considered significant, since earlier observers[2-4] had not been able to produce it in monkeys and apes after inoculating human trachoma tissues. Noguchi also stated[2] that the organisms could be isolated from the affected animals, and bacilli resembling them could be seen in stained film preparations of human and monkey trachomatous tissues.

A number of observers in different parts of the world have reported, during the past two years, confirmation of Noguchi's findings. In the United States specifically pathogenic strains of *Bacterium granulosis* were isolated from cases of trachoma in man by Finnoff and Thygeson,[5] in Denver, Colo.; by Tilden and Tyler,[6] in Arizona; by Olitsky and Tyler,[7] in New York, and by Kendall,[8] in Chicago. In the old world, Addario[9] recovered the microorganism from cases of trachoma in Italy and believed he had induced in man characteristic trachoma with a culture received from the Rockefeller Institute. Addario's culture was tested at the Institute and was found there to be active in a rhesus' conjunctiva, producing granular conjunctivitis. Stepanowa and Azarowa[10] isolated the microorganism specifically pathogenic for monkeys, from cases of trachoma in Russia. Weiss[11] found an organism corresponding to *Bacterium granulosis* in a patient at Tunis, which "produced granulations on the upper tarsal conjunctivæ and in the cul de sacs" of two Algerian magots. Recently Lumbroso[12] reported the cultivation of a similar bacterium from five out of seven Tunisians having trachoma and the production, with a culture, of characteristic granular conjunctivitis in a magot. That successful cultivation is not easy is, however, shown by the failures cited by Weiss.[11]

Certain previously undescribed properties of the microorganism are recorded in the articles by Finnoff and Thygeson[5] and by Tilden and Tyler.[13] For example, after prolonged artificial cultivation, but not after recent isolation, the bacterium may grow in

dextrose broth or on nutrient agar, though the growth is more abundant if Noguchi's carbohydrate mixture[2] plus horse blood or rabbit hemoglobin has been added.[13] It has been determined that *Bacterium granulosis* is Gram-negative (as opposed to the finding of Morax[4]); that in culture media indol is not formed; that nitrates are reduced to nitrites, and gelatin is not liquefied.[5 13] Other properties reported concern the prolonged viability of the organism in leptospira medium, at least six months; its sensitiveness to heat, 10 minutes at 57° C. being sufficient to kill it, and the harmful effects of cocaine on its growth.[5 13] The last fact is important in view of the common use of cocaine for anesthesia; it may explain the negative results of cultivation experiments by some observers.

In respect to animal inoculation experiments, the recent reports reveal that *Bacterium granulosis* could be maintained in culture and in monkeys for three years and could then induce in the rhesus the experimental disease,[13] and that, moreover, a pathogenic strain could be recovered from the lesions of a chimpanzee three years after inoculation. In connection with human trachoma, the recent article by Finnoff and Thygeson[5] states that *Bacterium granulosis* is not found in non-trachomatous diseases of the conjunctivæ and that, from the standpoint of diagnosis, the finding of the organism is more characteristic of trachoma than is the presence of inclusion bodies, because it can be isolated in the later stages of the disease when inclusion bodies are usually absent.

RECENT INOCULATION EXPERIMENTS.

The work of Noguchi has been elaborated by transmission experiments with material derived from trachoma patients and with cultures of *Bacterium granulosis*. The tests were done with the collaboration of Mr. Tyler and the materials from patients were obtained through the effective cooperation of Dr. Martin Cohen, to whom we are grateful for many kindnesses.

One series of tests made by Noguchi required further investigation since he reported[2] that he was unsuccessful in inducing the experimental disease in four rhesus monkeys and two apes inoculated with human materials. This led to an assumption by others that except with cultures Noguchi was definitely unsuccessful in inducing experimental trachoma. The assumption rests on insecure grounds, for Noguchi made only two tests on six

animals under unfavorable conditions: in one of the experiments the human tissue was kept nine days *in vitro* before inoculation.

We,[7] continuing Noguchi's work, have induced the experimental disease in monkeys by repeated swabbing with conjunctival secretions, and by a single injection of a suspension of tissues from human trachomatous cases. Indeed, *Bacterium granulosis* was isolated not only from the human material inoculated into monkeys, but also from the conjunctivæ of such affected animals.

Other transmission experiments relate to different modes of conveying infection to the conjunctivæ of monkeys.

Conveyance by Contact.—Noguchi[2] described the progression of lesions in monkeys injected with *Bacterium granulosis* as proceeding from the upper to the lower eyelid of the inoculated eye, and then to the conjunctiva of the uninoculated eye. The inference is that after infection occurs, the later processes develop from contact with the lesions or their secretions.

In two experiments, of which descriptions have been published,[1] the simple caging together of four monkeys with clear conjunctivæ and four animals showing granular conjunctivitis induced by *Bacterium granulosis*, brought about experimental trachoma in all the exposed monkeys. Finnoff and Thygeson[3] have recently confirmed the finding that monkeys may be infected by contact.

Conveyance by Swabbing Secretions.—We[7] have also noted that experimental trachomatous conjunctivitis is induced when secretions from monkeys showing *Bacterium granulosis* conjunctivitis are taken on cotton swabs and transferred on each of six consecutive days, directly, by rubbing, to the clear conjunctivæ of macaques.

Conveyance by Swabbing Cultures.—In a similar experiment,[1,7] the daily swabbing of the conjunctivæ with cultures of the microorganism, instead of secretions, also induced the experimental disease in monkeys. Under identical conditions, saline solution of uninoculated leptospira medium proved innocuous.

Conveyance by Instillation of Cultures or Tissues.—We[1,7] have shown, in addition, that the instillation of cultures into the conjunctival sac of normal rhesus monkeys on each of eight consecutive days, followed by gentle rubbing of the eyelids after each instillation, gives rise to characteristic granular conjunctivitis. On the other hand, in parallel experiments[1] in which the daily instillation was not followed by massage of the eyelids, the

monkeys were not affected. By way of contrast, however, the repeated daily instillations of suspensions of conjunctival tissue freshly removed from lids of animals exhibiting *Bacterium granulosis* conjunctivitis, induced in normal rhesus characteristic experimental trachoma. It appears, therefore, that some injury, even of slight degree—such as may be produced by rubbing the lids or by the presence of bits of tissue in the conjunctival sac—is necessary before successful implantation of the microorganism may be attained.

In planning the foregoing experiments, we tried to imitate the way in which the incitant of trachoma might be implanted on the conjunctiva of man, that is, by contact or by rubbing the eyelids.[4] The results of the tests indicate that *Bacterium granulosis* infection of monkeys can be secured by both means.

Conveyance of Infection with Human Materials.—We[14] have found that monkeys can be infected with the conjunctival secretions or tissues of human cases by repeated daily swabbing with the secretions and by single subconjunctival injection of the tissue. The details of the tests have already been described.[1,11] Although direct transmission of trachoma from man to monkey has been accomplished by several investigators,[2,4,11] the significant results of these experiments are the successful conveyance of infection to monkeys with, and the isolation of *Bacterium granulosis* from the same trachomatous material.

I may add here that *Bacterium granulosis* induces in monkeys granular conjunctivitis, which is indistinguishable from that caused by the inoculation of human trachomatous tissues. Several ophthalmologists have examined the eyes of the affected animals, and some have called the condition experimental folliculosis, others trachoma. It is important to note that the material injected into the conjunctivæ of the monkeys is derived from characteristic cases of human trachoma and of no other disease. It is therefore paradoxical to conceive that the animals respond to the inoculation of trachomatous material with a different disease.

SEROLOGICAL STUDIES.

The serological reactions of *Bacterium granulosis* are being investigated by Dr. Tilden. Preliminary experiments with rabbit immune sera prepared by the injection of cultures grown on semisolid medium appeared to indicate that the antigenic properties of *Bacterium granulosis* were rather weak and irregular.

One rabbit, after eight injections, seven intravenous (2-3 cc.) and one intraperitoneal (10 cc.), yielded a serum which had a titer of only 1:1. Another serum, after thirty injections of 5-6 cc., twenty intravenous and ten intraperitoneal, had a titer of 1:64. These sera were two years old when tested, but fresh pooled sera, similarly prepared, had a titer of only 1:16. It has proved possible, however, to prepare highly potent immune sera by injecting rabbits intravenously at five to six intervals with five gradually increasing doses (1 cc. to 5 cc.) of heavy suspensions of *Bacterium granulosis* grown on freshly prepared nutrient agar in Blake bottles. Such sera have agglutinated fourteen strains of *Bacterium granulosis* so far tested in dilutions of 1:256 to 1:1024 and are highly specific, having caused no agglutination of the common bacteria found in the conjunctival secretions or tissue of man or monkey (*M. albus, M. aureus, B. xerosis, B. influenzae*), or of numerous Gram-negative bacteria from the same source.

It is important to note, however, that the sera of sixteen monkeys which had developed marked granular lesions after subconjunctival inoculation of the organism have caused agglutination in one instance only, that of a rhesus which had had five intravenous injections of cultures during the month prior to the subconjunctival inoculation. The titre of this monkey's serum was 1:2. This fact may be correlated with the results of tests on sera from 24 cases of human trachoma in which no agglutinins for the microorganism were found. Complement fixation tests are so far indecisive, inasmuch as an antigen of sufficiently low anticomplementary titre and high specific binding power has not as yet been obtained.

INCLUSION BODIES.

The significance of inclusion bodies in trachoma has been discussed elsewhere,[1] the conclusion reached being that the corpuscles hitherto described are neither specific nor characteristic of typical inclusion bodies, such as are found in many filterable virus diseases. In a more recent summary of the subject, Weiss[11] cites an extensive literature that maintains this consensus of opinion.

Nicolle, Cuénod and Blaizot[15] advanced the hypothesis, which has not been confirmed by others,[11] that trachoma is caused by a filterable virus. Cuénod and Nataf,[4] in reviewing the work

of Nicolle and his coworkers, state that the "virus" is rapidly destroyed by heating at 50° C., by desiccation, and in eight days by glycerol. These are not the usual properties of true filterable viruses. Moreover, Cuénod and Nataf believe that the results of filtration experiments do not eliminate from consideration a "morphological microbic agent microscopically visible." This opinion is supported by the fact that thus far Dr. Knutti of our laboratory has not found in the materials derived from experimental and human trachoma any such inclusion bodies, cytoplasmic or intranuclear, as are characteristic of many filterable virus diseases.

SPONTANEOUS FOLLICULOSIS IN MONKEYS.

The occurrence of spontaneous follicular conjunctivitis in monkeys has been noted by Nicolle and Lumbroso,[16] and Wilson[17] believes that in most cases the inflammation is that of a simple folliculosis and certainly not of trachoma. However, this may be, it is generally agreed by investigators that the spontaneous affection should not be confused with experimental trachoma.

With regard to inoculation of trachomatous material, one should adhere to Nicolle's principle[18] of carefully examining animals and employing only such as have smooth conjunctivæ for proper interpretation of experimental procedures.

The problem of folliculosis in monkeys is now receiving our attention. So far no monkey of our normal stock has developed the spontaneous disease during a long period of observation. We have noted that in a certain dealer's stock a varying number of monkeys are affected at different times. A recent inspection of over 250 monkeys, nevertheless, kept at the same and at other dealers', failed to disclose any case of spontaneous folliculosis. Furthermore, *Bacterium granulosis* was recovered from none of eleven monkeys showing the disease. Finally, as has been already described,[1] cross-immunity does not appear to exist between the spontaneous granular condition and experimental trachomatous conjunctivitis.

SUMMARY AND CONCLUSIONS.

From the work of Noguchi and the later experiments of ourselves and others elaborating his findings, it appears that *Bacterium granulosis* is a newly discovered species of microorganism which thus far has been recovered only in human trachoma and

in experimental trachomatous conjunctivitis of monkeys and apes induced by the inoculation of cultures of the bacterium or of human material containing the organism.

The microorganism produces in monkeys and apes a chronic granular conjunctivitis which closely resembles trachoma in man. The infection of monkeys by *Bacterium granulosis* can be secured in several different ways. One is by transfer of the disease from an infected to an uninfected eye in the same animal, and another by caging together infected animals with monkeys having smooth conjunctivæ. These modes of infection are the counterpart of the process occurring during the evolution of human trachoma. Still other methods of conveying the infection are repeated transfer, by swabbing, of the conjunctival secretions of affected monkeys to the conjunctivæ of normal animals and repeated instillations of cultures followed by rubbing the eyelids or simple repeated instillation of suspensions of affected tissues of monkeys. Finally, the experimental disease can be produced by single subconjunctival injection of cultures. It is important to note that pathogenic strains of *Bacterium granulosis* can be recovered from the granular conjunctivitis induced by any of the procedures.

Of equal significance is the fact that human trachoma can be transferred to monkeys by repeated swabbing with secretions and by single subconjunctival inoculation of suspensions of tissues. The clinical and pathological appearance of the experimental disease thus effected is identical with that of the disease produced by cultures of the microorganism. Furthermore, *Bacterium granulosis* can be cultivated from the same trachomatous material used for successful inoculation of monkeys and also from animals so infected.

In conclusion, the work reported by Noguchi and his followers indicates that there is a close relationship of *Bacterium granulosis* to human trachoma.

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STUDIES ON BACTERIUM GRANULOSIS IN RELATION TO TRACHOMA: ITS PATHOGENICITY FOR VARIOUS MONKEYS AND APES.

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A careful examination of the protocols of the earlier workers on the etiology of trachoma for data on the relative susceptibility of various species of apes and monkeys to infection with the "Virus,"† or infectious agent, of human trachoma, reveals a divergence of opinion. It is readily realized that many factors, such as the technic of inoculation, the freshness or virulence, and the dosage of the virus used in the transmission experiments, the individual resistance of the animal and other unknown factors determine the success or failure of a given inoculation.[1] Moreover, there are no definite criteria as to what experimentally reproducible changes in the conjunctiva of monkeys are to be regarded as equivalent to trachoma in man. Some authors[2,3,4] consider the presence of secretion, inflammation and of Prowazek-Halberstaedter epithelial-cell inclusions as the criteria for the diagnosis of trachoma; others[5,6] regard these findings as evidence of a secondary virus infection, and a third group[7,8,9] considers the follicles or granulations to be the specific lesions.

Noguchi's recent discovery[10] of a bacterium which can be artificially cultivated and which induces certain granular lesions in the conjunctiva of monkeys opened a new approach to this field of investigation. As will be seen from Table I, Noguchi and his associates, Tilden and Tyler,[11] worked with rhesus monkeys in numbers far exceeding any previously recorded in similar studies.

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†The term "virus" is here used in the French sense, signifying the infectious agent as contained in material removed from human lesions without reference to filterability.

TABLE I. SUMMARY[1] OF ROCKEFELLER INSTITUTE PROTOCOLS: REACTION OF CONJUNCTIVA OF RHESUS MONKEYS AND CHIMPANZEES TO INOCULATION WITH BACTERIUM GRANULOSIS (CULTURES AND CONJUNCTIVAL SUSPENSIONS)

Species of Animals Used	Total Number of Animals Used	Authors	Animals with Results Entirely Negative. Per cent	Animals[2] with Mild, Transient Lesions, Lasting Less than 8 mos. ±, +, ++, +++. Per cent	Animals with Active, Progressive Lesions Persisting 8 mos. or longer, ++++. Per cent	Period of Incubation in "positive" animals. Days
M. rhesus	66	Noguchi	30.3	45.4	24.3	11 to 140
	93	Tilden and Tyler	58.0	26.8	15.2	11 to 150
Total M. rhesus	159		46.0	34.5	19.0	
Chimpanzees	6	Noguchi	16.6	50.0	33.0	8 to 120
	4	Tilden and Tyler	25.0	50.0	25.0	16 to 66
Total Chimpanzees	10		20.0	50.0	30.0	

1. This summary was prepared by the writer from the original protocols of Noguchi and of Tilden and Tyler.[11]
2. A few of these animals died before the 8th month with mild or active lesions, the majority, however, survived and showed only mild or transient lesions lasting from 2 weeks to 4 months.

They succeeded in inducing active, progressive lesions which lasted eight months or longer, in the conjunctiva of 19 per cent of a total of 159 animals (M. rhesus) and transient lesions (granulations which receded before the eighth month) in an additional 34.5 per cent. Their results in ten chimpanzees were as follows: progressive lesions lasting eight months or longer in three of the animals, and transient lesions, receding before the eighth month, in five. Two of the ten chimpanzees failed to react. These statistics compare favorably with the findings of previous investigators working with fresh trachoma virus obtained directly from trachomatous lesions. However, the long periods of incubation (up to 150 days) reported in the above studies are in striking contrast to the very much shorter periods (two to twenty days) observed by other writers in direct inoculations from human trachoma into human subjects and various species of monkeys.[12 13 14 15 16]

During the past two years* we have repeated some phases of Noguchi's work as part of an investigation on the etiology of trachoma. We have worked with three strains of B. granulosis, kindly furnished us by Dr. Flexner of the Rockefeller Institute, and later with two others, one isolated by ourselves while working at the Pasteur Institute of Tunis,[17] and the other in our laboratory at Washington University. We employed 28 rhesus monkeys, 2 Macacus inuus (Algerian magots), 1 baboon, and 1 callitriche. These animals were kept in clean, galvanized metal cages provided with removable wire bottoms of large mesh to allow feces to fall through into removable metal drawers which contained wood shavings. The animals were given a well-balanced diet of fruits, vegetables, cooked cereals, and in addition cod liver oil two to three times each week.

Noguchi's technic of conjunctival injection was followed in every detail, as described in his monograph and as later amended in the notes sent by Dr. Flexner. The technic suggested by Olitsky[18] of massaging the monkeys' lids was also used in the latter part of the work. A few of the animals were injected two or three times at intervals of several months, whenever the first or second injections proved to be negative.

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*We wish to acknowledge our appreciation of the kindness of Parke-Davis & Co., and of Eli Lilly & Co., who supplied us with the horse blood and serum used in the preparation of our culture medium.

Several selected protocols as examples of the types of lesions induced are given below:

PROTOCOLS.

Series I. Inoculation with B. granulosis (strain Tunis).

Experiment 1.—An Algerian magot, species Macacus inuus, named "Washington," was injected at the Pasteur Institute in Tunis, July 4, 1929, with a freshly isolated strain. Only the right eye was injected with a 72-hour-old culture grown in leptospira broth, in the manner described by Noguchi. On the sixteenth day there were visible in the conjunctival cul-de-sacs and in the superior tarsi of both eyes a few fine scattered granulations which burst easily. Inflammation was present at the internal angles, also marked edema of the lids, but no secretion. On the thirty-third day both eyes showed granulations persisting on the superior and inferior palpebral conjunctivæ. The lesions began to regress on the fiftieth day and had disappeared on the ninetieth day after injection.

Experiment 2.—A second Algerian magot, named "Zena," was injected with the same culture, at the same time and in a similar manner. The results were essentially the same, excepting that only the inoculated eye presented a few granulations on the internal and external extremities of the upper lid on the thirty-third day. The final outcome was similar.

Experiment 3.—A young male chimpanzee, "Joe," was injected on August 15, 1930, with the same culture, after it had been transported to St. Louis and after frequent transplantation in leptospira broth. The injection was made into the right eye with a 40-hour growth in Rivers's hemopeptone water. The culture was injected subconjunctivally into the superior retrotarsal fold and into the superior bulbar and inferior palpebral conjunctivæ. The cornea was scarified with the hypodermic needle and the lids massaged, as suggested by Olitsky. On the fifth day after injection there was marked edema of the injected site. The left eye was normal. On the sixteenth day the right eye presented three or four small colorless follicles on the fold leading to the caruncle and mild inflammation. On the 29th day both eyes showed two tiny follicles on the same fold. The conjunctivæ of both eyes returned to normal on the forty-ninth day and remained so up to the one hundred twenty-fourth day, when the animal was used for another test.

Experiment 4.—On January 16, 1930, a mature, female baboon was injected subconjunctivally with a 48-hour growth of the same culture (Tunis 34), grown in leptospira broth. Both the upper and lower lids of the right eye were injected. On the ninth day there was some edema and a mass at the site of injection. The conjunctivæ of the left eye were slightly irritated. The lesions did not progress and on the eighty-fourth day both eyes were normal. The same strain was reinjected on April 11, 1930, with negative results.

Series II. Inoculation with B. granulosis (Strains "A" and "B," Rockefeller Institute).

Experiment 5.—Rhesus monkey No. 1, male, possibly four years old, was injected April 30, 1929, with B. granulosis "A" (first culture sent from Rockefeller Institute). A portion of the contents of the original vial was injected into the right eye. On the tenth day there was edema and hyperemia at the site of injection and on the conjunctiva of the lower lid. The first appearance of follicles (three to four in number) was noted on the seventy-first day, on the superior margin of the tarsus, in the form of a horizontal ridge. Congestion and inflammation were present. The uninoculated eye became involved on the seventy-eighth day and showed two small follicles on the superior edge of the tarsus. Moderate swelling and edema. The lesions regressed slowly, and on the one hundred sixty-seventh day both eyes were normal but for a mild congestion of the cul-de-sacs.

Experiment 6.—On October 3, 1929, the same baboon used in Experiment 4, whose eyes were now entirely normal, was injected in the other eye (left) with a four-day-old culture of B. granulosis, strain "B." On the sixteenth day there were visible three or four fine, somewhat transparent follicles in the upper cul-de-sac of the injected eye. The uninoculated eye presented a slight edema of the lower palpebral conjunctiva and four or five follicles in the superior cul-de-sac. The tarsi were clear. On the fifty-sixth day both eyes showed four tiny follicles on the fold of conjunctiva leading to the inner canthus and above the tarsus. The conjunctiva of the lower lid was normal. Both eyes had returned to normal on the eighty-sixth day.

Series III. Inoculation of chimpanzees with cultures of B. granulosis (strain "B," Rockefeller Institute).

Experiment 7.—A young female chimpanzee, named "Mary," was injected December 3, 1929, with a 72-hour-old culture of B. granulosis "B" grown in leptospira medium. (This was the second culture received from the Rockefeller Institute.) The right eye only was injected, using 5 per cent cocaine as an anesthetic; the technic of Noguchi was followed. On the fourteenth day a mass was still visible at the site of injection. There were mild edema and congestion, but no follicles and no hyperplasia of the conjunctiva. On the one hundred and eighth day the plica semilunaris of the inoculated eye presented one or two follicles and there was hyperemia of the inferior lid. The other eye remained normal. On the one hundred fifteenth day the follicles had disappeared.

Experiment 8.—Another young chimpanzee (a male named "Joe") was inoculated with the same culture at the same time and in a similar manner. The observations made on the fourteenth day were essentially the same as those made on "Mary." A few reddish follicles were first observed on the thirty-fourth day at the external angle of the eye on the surface of the tarsus. A few small discrete follicles were also present in the superior cul-de-sac and there was much congestion of the conjunctiva. These follicles persisted and were still visible on the ninety-third day, when the uninoculated eye first became involved, and presented two or three colorless follicles on the conjunctiva of the lower lid. The lesions now regressed and by the one hundred forty-fourth day both eyes had returned to normal.

Series IV. Inoculation of a chimpanzee with material obtained from active lesions in a case of human trachoma.

Experiment 9.—A young female chimpanzee, named "Annie," was injected December 19, 1929, with an emulsion of bits of tarsus, and bulbar and palpebral conjunctivæ as well as follicles excised from an old active case of trachoma with scars, pannus and Prowazek epithelial cell inclusions. This material was injected into the right eye under cocaine anesthesia in accordance with Noguchi's technic. The conjunctiva of the left eye was scarified with a hypodermic needle but no material was injected. On the fifth day there was no change visible in either eye. The next examination was made on the sixteenth day after injection, when the right eye presented a mucopurulent secretion of the lower lid, and especially of the fornix. The superior tarsal conjunctiva was red and a few follicles were present on the fold

of mucous membrane leading to the inner canthus. The inflammation was quite marked, and was even worse on the eighteenth day. At this time the mucous membrane of the lower lid was drawn completely into folds. The uninoculated (left) eye now was definitely involved and showed a hyperplasia and papillary hypertrophy of the conjunctiva of the lower lid, beginning with the lid margin. On the twenty-sixth day the inoculated (right) eye presented eight follicles in the lower fornix; the mucous membrane was thrown into folds and inflamed. A few follicles were visible at the upper edge of the tarsus of the upper lid. About the thirty-ninth day the entire surface of the lower conjunctiva of both eyes was thrown into folds and profusely covered with follicles. The capillaries were visible only at the edge of the lid. There were also several follicles over the plica semilunaris and all over the surface of the tarsus, especially at its superior border. The conjunctivæ appeared dusky, salmon colored. On the fifty-first day a Prowazek-Halberstaedter body was demonstrable in the conjunctival scraping of the right eye. The lesions continued to progress and on the one hundred twelfth day the cornea of the right eye was scarified by means of a hypodermic needle and the lids were massaged in an attempt to evoke pannus formation by such irritation. The examinations made on the one hundred forty-fifth, one hundred seventy-sixth and one hundred ninety-sixth days after inoculation showed that the lesions had become quiescent. The final examination was made July 31, 1930, six and one-half months after inoculation. There were visible at this time a number of discrete, sagolike follicles, 2 mm. in size, on the superior and inferior palpebral conjunctivæ of both eyes, in the fornices, and on the surfaces of the upper tarsi. Some fibrinous secretion was present. The follicles in the right eye seemed somewhat vesicular and had a glassy appearance. No signs of pannus were observed in either eye. The animal died August 20, 1930 (the two hundred sixteenth day of the experiment). A pathological examination, made by Dr. McCordock of the Department of Pathology, Washington University, revealed pulmonary and visceral tuberculosis, intestinal intussusception, peritonitis and rupture of the tuberculous process into the bronchus. Histological studies of the eye are given in the text.

An examination of these protocols shows that various strains of Bacterium granulosis were capable of inducing in some mon-

keys a type of granular conjunctivitis similar to that described by Noguchi. The lesions in our animals, however, were all of the mild and transient type—i. e., never very extensive and never persisting more than four months. Varying degrees of edema and inflammation accompanied these follicles or granulations, but no thickening or loss of transparency of the conjunctiva or evidence of secretion were observed. The results may be summarized as follows:

TABLE II. RESULTS OF INJECTION OF VARIOUS MONKEYS AND CHIMPANZEES WITH CULTURES OF BACTERIUM GRANULOSIS

WASHINGTON UNIVERSITY SERIES.

Strain of B. granulosis Injected	Species of Animals Used	Number of Injections Made	Animals with Results entirely Negative	Animals with Transient Lesions Persisting up to 4 mo. (Receded)
Rockefeller "A"	M. rhesus*	3	2	1
	Baboon	1	0	1
	Callitriche	1	1	0
Rockefeller "B"	M. rhesus**	11	8	3
	Baboon	1	0	1
	Chimpanzee	2	0	2
Rockefeller M. C.	M. rhesus	5	4	1
	Chimpanzee	1	1	0
Tunis 34	M. rhesus*	5	5	0
	Baboon	1	1	0
	M. inuus	2	0	2
	Chimpanzee	1	0	1
St. Louis 13 L	M. rhesus	5	2	3
	Chimpanzee	1	0	1
Denver 7****	M. rhesus	3	3	0
	M. rhesus	32	24	8 or 25%***
	Baboon	3	1	2 or 66.6%
	M. inuus	2	0	2 or 100%
	Chimpanzee	5	1	4 or 80%
	Callitriche	1	1	0

Total Number of Animals Used:
28 M. rhesus (3 reinjected)
1 Baboon (injected 3 times)
2 M. inuus
2 Chimpanzees (1 injected twice, the other 3 times)
1 Callitriche

* Infection attempted twice.
** Infection attempted three times.
*** Calculated on the basis of total number of attempts at infection.
****Furnished by Drs. Finnoff and Thygeson.[22]

In a series of 28 rhesus monkeys (3 of them being injected more than once, making 32 attempts in all) there were 8 successful infections. Two Algerian magots, similarly injected, developed granular lesions. One baboon and one callitriche were also used. The baboon was injected three times and showed transient follicles twice. The callitriche was injected once but the result was negative.

In view of the fact that it has been suggested in the literature[19] [20] that chimpanzees are more susceptible to inoculation with material from human trachoma, three young chimpanzees were employed in transmission experiments. Two of these, named "Joe" and "Mary," were injected with cultures of B. granulosis (strain "B," Rockefeller Institute), which had been grown for seventy-two hours in leptospira broth. A third, "Annie," was injected in a similar manner with an emulsion of bits of tissue (tarsus, conjunctiva and follicles) freshly removed from a patient with chronic, active trachoma, showing scars, pannus and Prowazek inclusion bodies. The details are given in Protocols 7 to 9.

It will be seen from these protocols that the result obtained with the fresh trachomatous material was strikingly different from that obtained with cultures of B. granulosis. The animals injected with B. granulosis developed a few small transient follicles on the tarsal and palpebral conjunctivæ of the upper lids, while the one injected with the human material presented a picture which clinically and histologically showed a much stronger resemblance to human trachoma. The gross appearance of the lesions of this chimpanzee parallels closely the description of experimental trachoma induced by Taborisky[21] in human subjects.

It is of interest to note in this connection that attempts to isolate B. granulosis from the material removed from the conjunctival lesions of the patient as well as from those of the chimpanzee were unsuccessful.

Chimpanzee "Annie" unfortunately died on the two hundred sixteenth day of the experiment from intestinal and pulmonary tuberculosis. The histological sections of the conjunctiva show what many authorities accept as a typical picture of human trachoma. We are indebted to Dr. Harvey D. Lamb, assistant professor of ophthalmic pathology, Washington University, for the following report:

"Chimpanzee 'Annie.' Histological report on eyelids obtained postmortem:

Upper R. eyelid (the injected lid). The conjunctival surface is finely roughened with small areas of papillary hypertrophy and many shallow epithelial invaginations having an underlying slight infiltration with small lymphocytes and plasma cells; many young fibroblasts and new connective tissue, containing many small capillaries, are seen. Near the upper end of the tarsus the layer of infiltrated inflammatory cells becomes thicker and plasma cells and histiocytes are added.

"Lower R. eyelid. The conjunctival surface near the lids is roughened and shows epithelial invaginations, some of which are very deep. All this area shows a thin layer of dense infiltration with small lymphocytes, plasma cells and plasmacytoid cells (A. Fuchs) with *young fibroblasts* and histiocytes of endothelial origin. Around the deeper invaginations, in places, this infiltration becomes very dense and the formation of follicles with centers of epithelioid cells is seen.

"Upper L. eyelid. Same as Upper R. eyelid.

"Lower L. eyelid. The conjunctival surface is smooth. The covering is very thin, in many places but one layer thick, and in some places there is none at all. A layer of varying thickness of dense infiltration is seen just beneath the covering epithelium. This infiltration occurs in small foci and in some places small follicles are seen. The infiltration is composed of small lymphocytes with a very few plasma cells. The centers of the follicles contain groups of epithelioid cells. It may be noted in this connection that Axenfeld[1] and Lindner[3] stress proliferation and invagination of epithelium as important in differentiating trachoma from follicular conjunctivitis."

SUMMARY AND CONCLUSIONS.

1. Working with three strains of B. granulosis provided by the Rockefeller Institute, and later with two others isolated by ourselves, we have been able to produce only a transient type of follicular conjunctivitis, lasting not more than four months, in various types of monkeys and apes. We employed 28 rhesus monkeys, two Macacus inuus (Algerian magots), one baboon, one callitriche and two chimpanzees. Of the 28 rhesus monkeys 8 developed granular conjunctivitis. The two Algerian magots

developed similar follicles. One baboon was injected three times and transient follicles were induced twice, while the callitriche, which was injected once, remained entirely negative.

2. In two young chimpanzees, inoculated with the Rockefeller cultures of Bacterium granulosis, only transient follicles were induced, whereas in a third young chimpanzee, infected with fresh "virus" obtained from human trachomatous eyes, advanced lesions developed, which contained Prowazek-Halberstaedter bodies, and both clinically and histologically showed a striking resemblance to the lesions of an active human trachoma. Thus, eighteen days after infection, the mucous membranes of the lower conjunctiva were thrown into folds, there was papillary hyperplasia and hypertrophy, secretion and ptosis of the upper lid. The infection spread spontaneously to the uninoculated eye and follicles developed in the upper and lower conjunctivæ of both eyes, including the tarsi and retrotarsal folds. Attempts to isolate B. granulosis from the material removed from the conjunctival lesions of the patient as well as from those of the chimpanzee were unsuccessful. Histological sections of the conjunctivæ of the inoculated eye of this chimpanzee, taken on the two hundred sixteenth day after injection (when the animal died of generalized tuberculosis), showed roughening of the conjunctival surfaces, epithelial invaginations, lymphocytic infiltration, young fibroblasts and new connective tissue containing many small capillaries. Near the upper end of the tarsus the layer of infiltrated inflammatory cells was thicker and contained plasma cells and histiocytes. The lids of the uninoculated eye showed a similar picture. The centers of the follicles contained groups of epithelioid cells.

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DISCUSSION.

Dr. Paul D. Mossman (U. S. Public Health Service, Albuquerque, New Mexico): I appreciate the privilege of listening to this symposium on a subject in which I am greatly interested. There are a great many angles to this disease and a great many points of interest. My work has been particularly in the line of prevention of the spread of the disease, and I am glad that Prof. de Grosz gave us so much on that particular phase of the problem. He spoke of the reporting of the disease. A good many states make the disease reportable, but we know from our investigations and experiences that there is a great deal more of it in this country than is reported, and it seems to me the members of this Academy might contribute quite a bit toward the prevention of this condition if they would take the trouble to report to the State Health Department the cases they find.

It is my belief that one of the most important factors in the relief of the individual cases of trachoma, as well as preventing complications and sequelae, lies in early diagnosis and treatment. I believe early treatment, even though not ideal nor adequate, very often prevents sequelae. I have seen a good many cases of long standing that give a history of having had some treatment in the early stages, and these cases very seldom present the distressing complications and sequelae, including damaged vision.

Dr. de Grosz gave us some enlightening figures as to the prevalence in various countries. Our percentage figures in this country are apt to be misleading. It is not uniformly spread over any one of our states, or over any county in any state; it exists in neighborhoods. In the mountain districts, to which Dr. Stucky has made such eloquent reference, you will find it in the shoestring communities—where the creek bed outlines the community and the people have social contact with the people up and down one valley, but not with people in another valley. You may find one valley in which trachoma is highly prevalent, and over in the next valley not so much. So if we quote figures on the prevalence we have to take them with a grain of salt.

One more point I would like to call attention to, and that is the point of pannus as a means of diagnosis. I would like to express my personal belief that pannus is of much earlier occurrence in trachoma than we used to think. If you will search for pannus with magnification and oblique illumination you will often find it earlier than you will without these aids. You will see a good many cases of folliculosis which show the vessels coming down over the limbus forming a curtain with a straight edge, but in early trachoma you will often see a few vessels down from the edge of the curtain giving an irregular margin. This I feel is diagnostic of trachoma.

Dr. William C. Finnoff, Denver: The two papers that I wish to discuss are those of Dr. Olitsky and Dr. Weiss. We reported the results of our experiments with *bacterium granulosis* at the meeting of the Section on Ophthalmology of the American Medical Association this year. In our paper we stated that we had confirmed in every detail the experimental work of Dr. Noguchi, and in addition we have isolated the organism from nine out of twelve white persons with unquestioned trachoma. All had

pannus, scar tissue, and unquestioned lesions of trachoma. The organisms were injected into the subconjunctival tissue of monkeys and in a large percent, larger, I think, than at the Rockefeller Institute, we succeeded in producing conjunctival granulosis. In some of them follicles have existed for a period of twenty months; in two, definite evidence of scar tissue occurred, but in not one has there been pannus. We recently sectioned one of the eyes and lids in which there was newly formed scar tissue. This showed a band of scar tissue and histologic evidence of dense scar tissue formation. Other sections have been made and active lesions show follicles that are identical in appearance with the follicles seen in human trachoma. There was also interfollicular infiltration—that is so characteristic of the lesions in trachoma.

We have also carried on serological studies. These experiments were done exclusively by Dr. Thygeson, and it was found that the experiments confirmed the results of the last two speakers in that none of the cases of trachoma shows evidence of agglutination. However, in rabbits that had been injected intravenously with bacillus granulosis, agglutinin was found in the serum. This agglutinin was specific for certain types of the bacillus, and suggests that there are probably different strains of *bacterium granulosis*. It is not surprising that we have negative agglutination in case of trachoma. The disease is confined to a small area in the conjunctiva and in some respects simulates disease of other epithelial structures which do not give positive serological findings. We think the serological proof of the presence or absence of trachoma will be difficult and will not be reliable as a diagnostic measure.

We have compared our cases of known lesions with three animals that we now have with spontaneous folliculosis, and the lesions produced by *bacterium granulosis* in our animals are very different from those of spontaneous folliculosis.

In the conclusion of our paper read in June we stated that a disease not unlike trachoma was produced in monkeys after the injection of *bacterium granulosis*, but the disease was not exactly similar to human trachoma, and in order to prove that *bacterium granulosis* is the cause of human trachoma it would be necessary to inject the human being.

LATTICE KERATITIS: STUDIES OF FOUR CASES OBSERVED IN ONE FAMILY.

ALLEN GREENWOOD, M. D.,

BOSTON.

I have chosen Lattice Keratitis for the title, as this most nearly describes the appearance of the corneal dystrophy as seen in three of my cases where the disease was in its earlier stages. The term nodular keratitis is often used for this rare disease of the cornea as well as nodular opacity, nodular degeneration, family punctate degeneration and grill-like keratitis. Professor Gallemaerts[1] labels the condition Keratite Grillagée in his monograph entitled "Examen Microscopique des Affections de la Cornée au moyen de la Lampe a Fente," presented to La Societe D'Ophtalmologie, May 10, 1926. See Planche X, Fig. 59, of his paper.

Goar[2] describes the corneal condition in his case, reported at the 1930 meeting of the American Ophthalmological Society, as follows: "The corneas had lost their luster and upon casual inspection looked as though they had been smeared with shellac. On close examination with the loupe and lens it was noted that the superficial part of the corneas were crisscrossed with interlacing lines, giving much the same appearance as a section of lung tissue under the microscope. Scattered through this latticework in an irregular manner were tiny nodules of a dirty gray or grayish-yellow color. With the slit-lamp it was noted that the larger of the areas contained as many as five nodules, while the smaller were single or in pairs. They were imbedded in the superficial part of the stroma and the grayish lines mentioned above were apparently in or just beneath Bowman's membrane. The whole surface of both corneas, except a narrow ring adjacent to the limbus was affected." Evidently from this description the lattice appearance was very apparent and was possibly more descriptive than the term nodular.

It seemed advisable to present this family, with five known afflicted members, as the disease was present and observable in so many stages.

Case 1.—This was the case of the mother of my other three patients. I saw this patient first June 19, 1925, in consultation with Dr. William F. O'Reilly of Lynn, Mass. She was 61 years old, born in Vilna, Russia, and gave the following history: Her mother and grandmother had both had failing vision beginning in early adult life and ending in practical blindness before they died. She herself began to lose vision in her right eye when about 30 years old, and soon after in the left eye. This progressed until the right eye was nearly blind. Recently she has been quite ill with gall bladder trouble, and during this time the left eye has been inflamed and painful and quite blind.

Examination of the right eye showed the cornea looking like a piece of old parchment with numerous specks of quite yellow material scattered through its substance. The surface was rough and nonlustrous and the central three-quarters of the cornea was

Case 1.

slightly conical. There was, however, a band of normal cornea about one mm. wide at the limbus; all around and through this a normal appearing iris could be seen. The conjunctiva was normal and there was no circumcorneal injection and no vessels seen on cornea. Vision equaled light perception.

Examination of the left eye, which had been until recently the better eye, showed two-thirds of the central cornea partly destroyed by a serpiginous ulcer, with a grayish infiltration surrounding the ulcer. The greatest depth of the ulcer was in the lower part. There was considerable circumcorneal injection. As the ulcer had been spreading, a complete covering with conjunctiva was advised after curetting the ulcer. This operation was performed by Dr. O'Reilly August 11, 1925, at the time of a gall bladder operation, with excellent results, for when the suture

holding the conjunctiva was released there was no active ulceration, but the cornea was flattened in appearance, there being no regeneration of the destroyed corneal tissue. The three mm. area around the old ulcer was clearer, especially above.

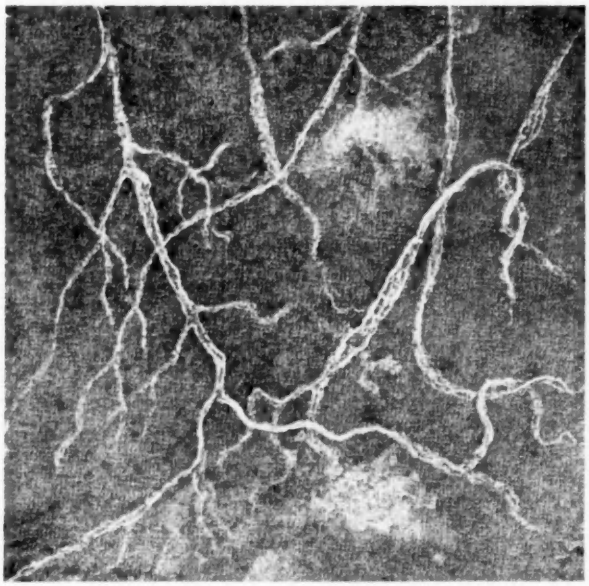
I have seen this patient from time to time since October, 1925. The right eye has shown no change except a slightly deeper yellow color to the plaques or nodes so thickly scattered throughout the degenerated corneal tissue. The rim of clear cornea is still not encroached upon, and with the pupil dilated light perception is a little clearer, though the lens as thus exposed is diffusely cloudy.

The cornea of the left eye, when last seen, February 25, 1930, appeared somewhat clearer and the facet on the central and lower part of the cornea not so deep. There was an anterior synechia at 6 o'clock. Around the gray depressed central portion deep interlacing lines of opacity could be detected. With the pupil dilated a cloudy lens could be seen and the vision equaled counting fingers at one foot. The narrow peripheral band of clear cornea was free of blood vessels. During the five years she has kept the vision of her left eye a little more useful by the daily application of some mydriatic, at times atropin in a 1 per cent nosophen ointment and again hyoscin hydrobrom 1/10 per cent in 1 per cent holocain solution. She has never developed an atropin poisoning.

This case illustrates the end-results of the familial dystrophy, here called lattice keratitis, the appearance of the left eye having been altered by the superimposed infected ulcer.

Case 2.—This patient was the daughter of the patient described in Case 1. Seen December 6, 1927, aged 39 years. For two years her right eye has been steadily losing vision and lately the left has been growing dim. Neither eye has ever been inflamed or painful. So far treatment has not helped. Her blood Wassermann was negative, she reports, but she had had antiluetic treatment. Examination shows a noninflamed right eye with a clear rim of cornea about two mm. wide, while the central portion of the cornea is extensively involved by a lattice arrangement of opaque bands with a few opaque spots near the crossing of the interlacing bands and between. These opacities appear to lie in the anterior layers of the stroma. In the center the surface of the cornea is slightly roughened in appearance. The left eye shows the same condition in less degree and is confined to the center of the cornea and extends slightly down and in. This area

is about four mm. in diameter and the surface of the cornea is clear and smooth. A —1.50sp = +3.50c. at 105° gives vision of 20/70. The patient was seen by Dr. George E. deSchweinitz in consultation. He confirmed the diagnosis and kindly called my attention to the striking similarity of the slit-lamp picture presented in this case and the slit-lamp picture used by Gallemaerts to illustrate Keratite Grillagée in his monograph. I had made a drawing of the slit-lamp appearance in the left eye of this case, but as that of Professor Gallemaerts was so much better I have adopted his for presentation here.



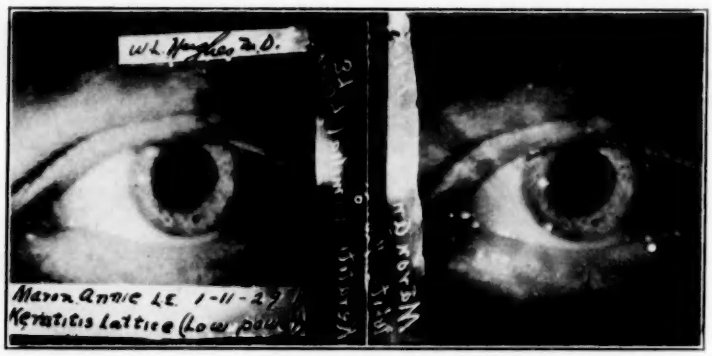
Gallemaerts' Case.

Thinking there might be a neurotrophic basis for the trouble, she was given a 1 per cent solution of holocain to use twice a day. In two months the vision in the left eye had improved to 20/40, much to the patient's satisfaction, though no change could be noticed in the corneal picture. She has used the holocain ever since and the vision is still 20/40.

At one time, following a prolonged slit-lamp examination, there developed intense photophobia and epiphora which was relieved by an ointment of nosophen. At one time, thinking some faulty

secretion might be present, thyroid extract was tried, but this was not well borne. On January 8, 1930, Dr. J. Herbert Waite made a careful slit-lamp examination and reported as follows:

Right Cornea.—Lattice pattern involves entire cornea, more dense centrally, where the corneal surface is roughened, and less dense peripherally. By slit-lamp there are two types of opacity in the cornea: 1. A *syncytial* net of well defined, *refractile cords* of unequal caliber, thicker here, thinner there, with terminations in *pointed tips.* The terminations are best seen around the periphery of the cornea, from one to two millimeters from the limbus, and never invading the clear belt of cornea at the limbus. Occasionally one finds an isolated cord, with branching and with pointed terminals, not visibly connected with the main syncytial net.



Case 3.

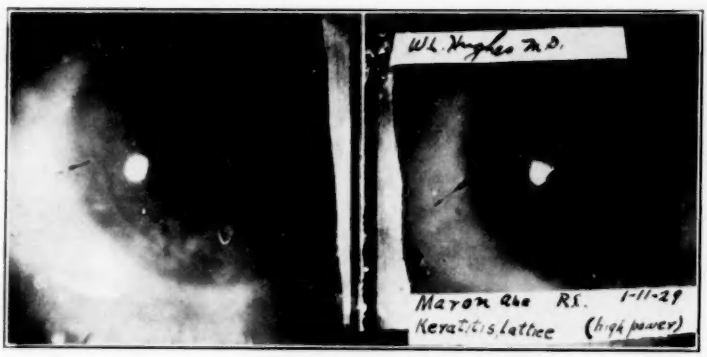
Depth: Over the central cornea one finds heavy cords at all levels of stroma, and from optical section one would gain the impression that the central cornea is thickened, perhaps 25 per cent. Both the posterior face and the anterior face of the central cornea is roughened; just temporal to the pupillary zone of the cornea and extending from the epithelial level to a depth of one-fourth of the total thickness of the cornea there is a cystic space filled with a light brownish fluid. The aperture of this space is oval and approximately .1 mm. wide. The depth of the refractile cords peripherally is invariably deep within the stroma, almost to the level of Descemet's membrane.

2. *Flocculent, nebulous clusters,* with ill defined edges, are seen at all levels of stroma, and particularly through the central

cornea. There are no cholesterin crystals in these flocculi or elsewhere in the cornea and *no blood vessels.*

Left Cornea.—Abnormalities mostly found in lower nasal quadrant, from pupillary zone outward. Few abnormalities are found in lower temporal quadrant.

1. Syncytial net of refractile cords less well developed, showing more isolated and unconnected branching forms. In only two places do the cords come forward to the level of Bowman's membrane. The terminations are pointed, and almost always in the



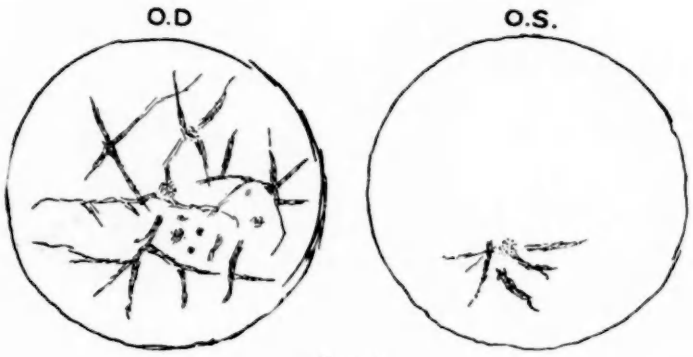
Case 4.

deep stroma, several millimeters from the limbus line. The cornea is not thickened, and no cystic spaces are to be seen. The upper portion of the cornea is clear.

2. Flocculi are present, but to a very much less well pronounced extent than in the right cornea.

No doubt one guess is as good as another, until one gets the actual tissue for histologic study. From the appearance of the syncytial net, I am tempted to think of this condition as a degeneration of the lamellæ of the stroma or proper substance of the cornea, with eventual destruction of Bowman's membrane centrally, and possibly even of Descemet's membrane. There is no vascularization to be made out in either cornea. The process undoubtedly begins (see O. S.) in isolated spots or islands, which later join to give a complete syncytial net of opacities in stroma. The degenerated tissue may after a long time undergo still further changes of hydropic nature.

Case 3.—This patient was an older daughter of the mother described in Case 1. Seen first March 22, 1928, aged 44 years. Right eye growing dim for many years and now nearly blind. Left eye recently failing. No help has been derived from the treatment of several oculists seen. Also, as in Case 2, a negative Wassermann was obtained. Examination shows the right eye as in Case 2, but farther advanced in that the center of the cornea shows a raised cone of dense opacity having a yellowish color and parchment like surface. Left eye almost identical in appearance with the left eye of Case 2, and vision equals 20/40.



Case 4.

Case 4.—The son of the patient described in Case 1 and the youngest of her afflicted children and the least affected. Seen first May 11, 1929, at age of 38 years. First noticed disturbed vision ten years ago, especially in right eye. Since then there have been periods of photophobia, and the vision of the right eye has grown slightly less. Examination shows right cornea quite similar to that of the left eye in the two preceding cases, while the left eye shows only one nodular spot with a few lattice bands.

Vision in right eye, 20/70; vision in left eye, 20/30. I saw this patient in my office only once.

Case 5.—A grandchild of the patient described in Case 1, not seen by the writer, but Dr. O'Reilly reports that both eyes show a typical lattice keratitis in the early stages.

Whether the mother and grandmother of the first patient also had lattice keratitis I cannot, of course, be sure, but from the meager description of their condition given me it seems quite probable. I cannot offer any explanation as to why the right eye

in all my cases was the first affected and became by far the most disturbed. None of my patients had any evidences of syphilis or tuberculosis. They are all stout and now in apparently good health. Patient 4 showed on blood examination an increase in cholesterin. Patient 2, tested for the same, showed no increase of cholesterin, so no abnormal physical condition was found in any of the patients to account for their trouble. Certainly they have nothing in their general condition or in their ocular manifestations to even suggest tuberculosis.

For this rare disease there is quite a voluminous bibliography, but as this has been so admirably covered by Dr. Goar in his very recent article it is not necessary for me to cover it here. I wish, however, to mention one of the most illuminating studies, namely, R. Stanka's[3] article on "Familial Grill-like Corneal Degeneration," describing the corneal changes in his case. He found "the two eyes to be entirely free from irritation. The surface of both corneas was smooth. The right cornea more distinctly than the left presented typical grill-like streaks, especially dense in the center, becoming thinner and scantier towards the periphery. In addition, the middle corneal portions presented masses of gray spots and dots. The corneal sensibility was greatly diminished in the middle portion on the right side, less so in the peripheral portions; on the left side it was normal. Examination with the slit-lamp showed in all layers of the corneal parenchyma irregular crenated spots, in part very indistinctly outlined; in the superficial layers, they were light gray, rather blurred, in the deeper layers denser and more sharply outlined, sometimes glistening like crystals in the vicinity of Descemet's membrane. These macular opacities do not always lie in the same plane, but also obliquely, passing through several corneal layers. The dense network of the so-called grill fibers is the most striking; these fibers are densest in the middle of the cornea, decreasing in density towards the periphery, tapering off and finally disappearing before the limbus is reached. Seen in direct light, these hyaline transparent structures are somewhat thinner and finer grayish-white streaks, situated in different layers of the cornea, being more superficial at the periphery than in the center."

On vital staining he apparently proved that the grillwork and nodules were not connected with the corneal nerves. His article in full should be read by those interested in this disease. Jacqueau,[4] in 1909, also wrote a good description of the grill-like

lines in the corneas of a family of eight that came under his observation.

From a study of the literature it is at once seen that one of the chief characteristics of lattice keratitis is its familial tendency. It is also always eventually bilateral and slowly progressive, being apparently uninfluenced by treatment, and there are usually no inflammatory signs unless some complication ensues. The end result is a parchment like cornea and vision of light perception only. No cause has as yet been determined. The most acceptable hypothesis apparently is that it is a form of hereditary degeneration or dystrophy of the whole structure of the cornea with deposits of nonassimilable material. This material is, according to Paderstein[5] and Wirth,[6] who examined specimens, hyaline.

Dr. Goar also made a histologic examination of a wedge of cornea from his case and describes the microscopic picture as follows:

"The epithelium is very irregular in contour, in some areas being thickened to twelve or more layers; in others, thinned to barely two or three. The thinning occurs over irregular spherical nodules which stain pink with eosin and yellowish with the indigo picric acid carmine stain. There is not much change in the epithelial cells except that the basal cells have lost their columnar aspect and individual cells are packed closely together. In some areas the cytoplasm appears vacuolated. No vestige of Bowman's membrane can be found. The stroma takes a heavy eosin stain and there is a marked tendency to splitting into lamellæ. The nodules which lie just beneath the epithelium are always separated from it by a thin layer of connective tissue. The nodules consist of an amorphous substance, staining yellow with the picric acid, and resembling hyaline. No granules of this substance are found in the epithelium."

Another theory is that the lesion begins in the nerves and nerve endings and is therefore neurotrophic, according to Kraupa,[7] Collins[8] and Pillat.[9] Stanka's case does not seem to bear this out. Others, like Wehrli,[10] have thought that the condition was a very inactive form of corneal tuberculosis, but in my cases this does not seem possible. It has been suggested to me by Dr. Parker Heath that this familial degenerative disease might have a lipoid basis and be due to some pituitary or thyroid fault, and the patients should be on a fat free diet plus endocrine therapy. A future study of histologic specimens when obtainable may throw more

light than we now have on this baffling condition and its causes, and thus possibly pave the way to a more successful therapy.

I want to take the opportunity to express my appreciation of the kindness of Dr. deSchweinitz, Dr. Goar and Dr. Waite in helping me to prepare this paper.

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DISCUSSION.

DR. RICHARD C. GAMBLE, Chicago: Dr. Greenwood's observations of this group of patients of various ages, with lattice keratitis in various stages, is of great value because it is almost the same as though he had observed a single case through its entire course. It is possible in such a group study to come to certain conclusions with a greater degree of accuracy than would be possible from a most detailed examination of a single case over a period of only a few years.

Certain facts stand out in this report which impressed me as being of great importance in our understanding of this disease. The grill-like bands were found within the substantia propria of the cornea, and in the earlier cases the superficial layers were normal. The superficial changes in the cornea which were found in the older patients, the rough, nonlustrous surface and the parchment-like appearance may be entirely secondary to the primary change in the stroma. These superficial changes were those most dwelt upon in the cases reported before the advent of the slit-lamp, and were somewhat confused with the lattice-like bands due to inability to determine accurately the depth of the opacity with the instruments then in use. Apparently certain theories must be discarded, for example, the conception that the lattice bands are due to wrinkling of Bowman's membrane due to low intraocular tension. The bands are not in Bowman's membrane, and eyes with low tension are not especially prone to develop lattice keratitis.

We know this disease is familial; we have every reason to consider it a degenerative condition, probably a form of abiotrophy rather than an inflammation, and we have good reason to believe that it affects, first, the tissues between Bowman's and Descemet's membranes. That narrows down the possibilities considerably.

The theory that this disease is neurotrophic in origin cannot easily be discarded. The caliber of the opaque bands, the type of branching which they make, and the fact that they seem in some cases to interweave so that the same band will occupy different levels seem to indicate that the nerve fibers become opaque. Also, the clear zone at the limbus may be due to the fact that some of the corneal nerves are medullated a short distance into the cornea. The fact that the more peripheral opacities are deeper than the central ones is of some importance. The presence of corneal anesthesia in some cases is suggestive, but might be due, secondarily, to changes in the cornea through which the nerves pass.

Dr. Waite concluded that the opaque bands are due to degeneration of the corneal lamellæ. His description is very clear, but it gave me a somewhat different idea, namely, that the opaque bands, or as he describes them, the refractile cords, might be due to some form of hydropic degeneration of the fixed corneal corpuscles. When they are examined, not by cross-section, but by flat-section, they are seen to be composed of cell bodies with long processes forming a syncytium extending between the corneal lamellæ. Perhaps the nebulous clusters are due to a change in the cell body and the refractile cords to involvement of the processes.

Dr. Goar, in his histologic report, states that the corneal stroma takes a heavy eosin stain, but that there is a marked tendency for the cornea to split into lamellæ. This is suggestive. This idea is offered purely as a speculation and with some apology, as there are already many speculations concerning this disease.

THE BARRAQUER CATARACT OPERATION (MODIFIED).

OTIS R. WOLFE, M. D.,

MARSHALLTOWN, IOWA.

An increasing number of ophthalmic surgeons are turning to some form of intracapsular operation or adopting some procedure to do away with the capsule at the time of the extraction.

The erisiphake method was first described by Hulen in 1910, but was popularized by Barraquer. A. S. and L. D. Green[1] modified the apparatus and also the technic by combining expression with it. Barraquer now uses more or less expression with most of his cases.

I presented my technic and arguments in favor of the Barraquer-Green operation before the eye section of the Iowa State Society[2] in May, 1925, and another article dealing with its advantages in soft cataract in 1927.[3] The technic I am now illustrating is the same with slight variations. It is a modification of the technics of Barraquer and Green.

Focal infections are carefully searched for and eliminated when found. Blood sugar and blood urea are estimated and both reduced if high. Elimination is raised as high as possible. Tension is taken, and if above 28 mm. operation is deferred and treatment instituted or a preliminary iridectomy performed. Silvol or argyrol and bichlorid ointment, 1–1,000, are instilled every one or two hours for at least twenty-four hours previous to the operation. Bromides or other sedatives are given the night before and immediately preceding the operation. Care is taken to calm the patient and to teach him deliberate and easy closing of the eyes. He is impressed with the fact that the operation is to be painless and should he feel pain that he is to calmly notify the operator and that the operation will be stopped and more anesthetic used. The morale of the patient is most important. Nothing is more desirable to the surgeon than a placid patient.

Exacting technic is accomplished in our practice by having a cataract operating team of five members, each member being trained to care for certain details. Highly skilled and trained

assistance is absolutely necessary. The operator himself tests and checks the apparatus. The first assistant controls the lids, watches the sutures and performs other steps without instruction. It is absolutely essential that he do certain steps without delay or direction. The same nurses assist each time and are familiar with every step of the operation. One assistant stands behind the operator, hands and takes the erisiphake out of his hand and controls the foot valve. The instrument nurse wears gloves and care is taken not to touch the part of the instruments entering the eye. A preliminary iridectomy is performed, in the majority of cases, a week or ten days before the extraction. The pupil must be kept well dilated so that synechiæ do not form. It is much easier to make a good incision and a broad conjunctival

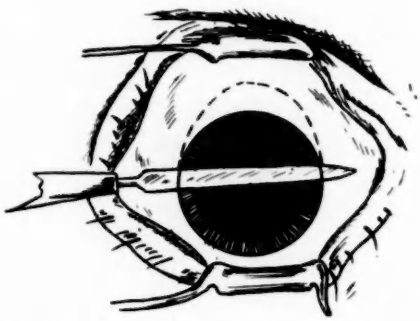

Fig. 1. Full one-half section of cornea. Knife enters and emerges at limbus. Large conjunctival flap. Large preliminary iridectomy.

flap where a large preliminary iridectomy has been performed. The knife then never engages the iris at the upper angle, even if the anterior chamber is shallow. Wide dilatation is often not obtained in elderly people with cataract.

Five per cent cocain is instilled and the lids injected with 2 per cent novocain. The speculum is introduced, and if the eye is small or deep seated a full canthotomy is performed. A few drops of one-half per cent cocain are injected under the superior rectus and a heavy control suture inserted as described by McReynolds.[4] I agree with him fully as to its value. If this is placed far up a lid hook is not necessary, for the upper lid and the lower lid is controlled by the thumb of the assistant or by a stitch. It is absolutely necessary to make a full one-half section of cornea (Fig. 1). One or two drops of 5 per cent cocain are

injected into the anterior chamber. If a preliminary iridectomy has not been performed, it can be done at this time. A suture of fine Kalt silk in a Barraquer needle is placed through and through with a loup of about three-fourths of an inch laid to one side. The ends of the suture are tied and laid to one side so they can be quickly picked up. The speculum is then removed.

The second assistant hands the canula to the operator over the right shoulder and watches the tubing to see that it does not bind or pull. He also takes it from the operator's hand without further instruction after the cataract is removed. The ball tip forceps de-

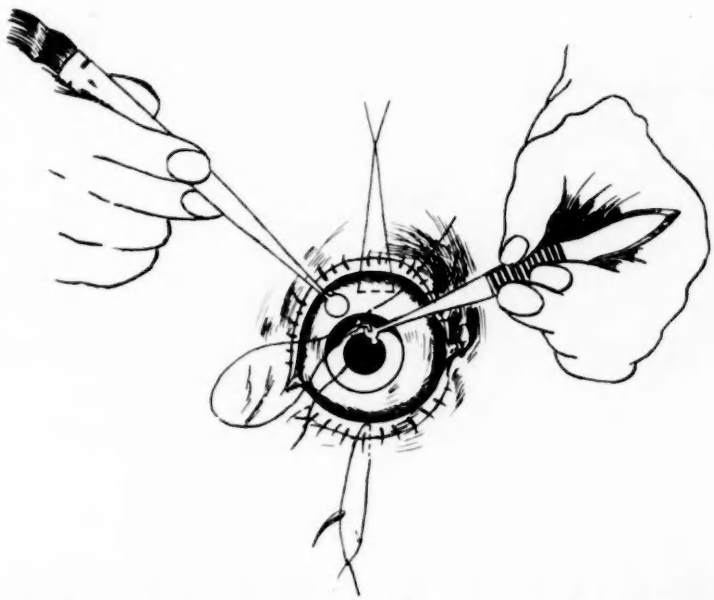

Fig. 2. Corneal section grasped by forceps, turned back. Canula placed on lens by direct view.

scribed before this society at the 1928 meeting[5] is held in the left hand throughout the operation. The corneal section is grasped with the forceps and turned back (Fig. 2). The canula is placed exactly on the capsule by direct view and not watched through the cornea. By so doing there is no excuse for catching the iris margins. If the pupil has not responded well to dilatation, a smaller canula is used. The large canula is preferable, and it is much easier to use where a preliminary iridectomy has been per-

formed. There is less blood in the anterior chamber and the detail is much better.

The operator wears a Beebe loup and the attachment can be accurately observed as the vacuum is applied. One of the assistants reads aloud the amount of vacuum attained. It is varied for different cases. Barraquer uses 55 mm. Hg. for soft cataracts and 58 mm. for the others. The canula is then rocked gently from side to side a few times to rupture the suspensory ligament. This rocking also does away with the temporary vacuum produced behind the lens which would tend to lift the vitreous as well. The corneal section is then released by the forceps and they are trans-

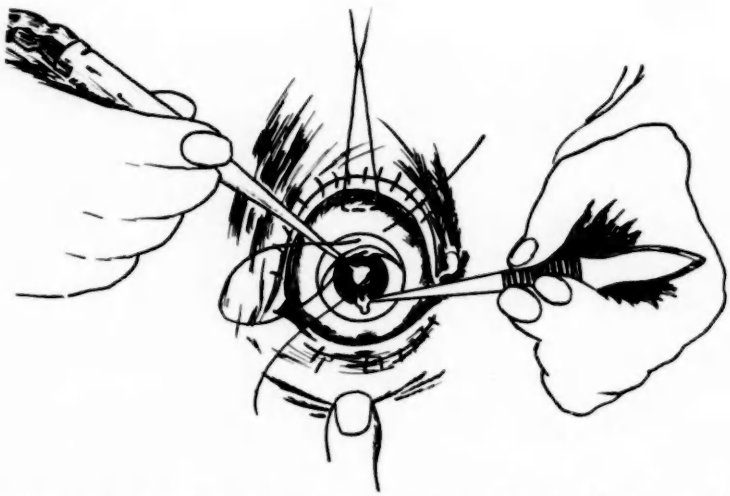

Fig. 3. After canula attaches well to capsule, forceps closed, transferred to limbus externally. Ball tip makes counter pressure.

ferred to the globe (Fig. 3). The ball tip makes counter pressure at the limbus. The amount must be varied to suit the case. As a rule, the younger or better developed patients require considerable counter pressure. When pressure is first applied, it may be alternated with relaxation, but once the lens starts to dislocate from the patellar fossa it should be firmly followed as in the Smith technic. If too much lift is made on the canula without counter pressure the capsule will rupture, but seldom before it starts through the wound. When it does this, the capsule and lens débris deliver so that they seldom require any attention whatever.

It accomplishes the same result as an intracapsular extraction. When the proper amount of expression and lift are combined the lens will usually appear, upper border first, and then be lifted out by the canula without further counter pressure. Occasionally the additional lift with less expression causes the erisiphake to lose its hold on the capsule before delivery is complete. If so, the first assistant has a Fisher needle ready and assists the lens through the wound. This is more desirable than following the lens with too much expression. If the capsule ruptures, the use of the erisiphake is immediately discontinued and the lens is expressed by counter pressure exactly as in the capsulotomy method. A modified Snellen loup is used if vitreous presents before delivery is commenced.

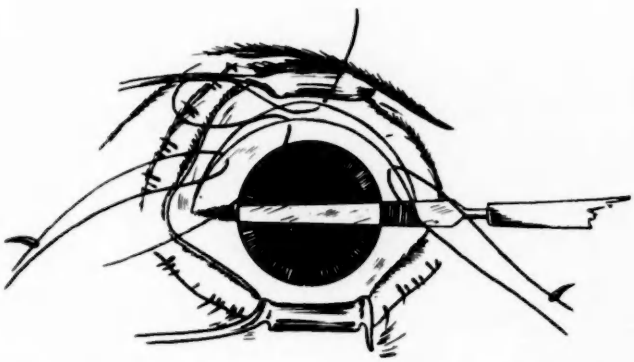

Fig. 4. Large circular conjunctival flap dissected to limbus. Sutures hold flap up as incision is made. Extraction and central stitch as in Figures *2* and *3*.

When the pupil is widely dilated and the canula can be placed well down on the lens, it can be tumbled advantageously, as advocated by Cruickshank.[6] The suture is quickly pulled taut and cut, and the eye immediately closed for ten to fifteen minutes. This is a very important step. Too much zealousness or toilet at this stage may be very harmful. If a bead of vitreous presents, it will usually drop back. Vitreous does not run out. It is either pressed out by the surgeon or the patient. If the lens has been removed in the capsule, no toilet is necessary except smoothing out the conjunctival flap, after ten or fifteen minutes have elapsed, and instilling the bichloride ointment.

Barraquer does a marginal iridectomy after the extraction to prevent iris prolapse. We prefer a large idirectomy previous to extraction and no iris prolapse can occur.

Figure four illustrates a variation in the technic that we have used in obstreperous patients or those who cannot lie on their backs longer than a very few hours or in cataract with glaucoma. The first step in the operation is to dissect a large circular conjunctival flap from above and on the sides. Two sutures are placed on the sides. They are held up alternately like a tent by the assistant, while the incision is made underneath them. The threads are not in the operator's way as a forceps would be. The knife comes out above in the sclera in direct view as the flap is held back. The needles are left on the thread of the lateral

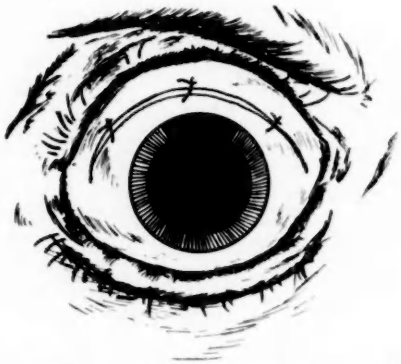

Fig. 5. After extraction central suture draws flap together. Needles left on lateral sutures Fig. 4 passed through conjunctiva above, section closed. Additional sutures or water tight suture used in glaucoma.

sutures and laid to one side. One central stitch is placed as in the original operation. The balance of the technic is as above described. The central suture is drawn first and tied. The needles of the lateral sutures are then passed through the bulbar conjunctiva above and tied, making a complete closure of the wound (Fig. 5). Patients can sit up in bed after two to four hours. One patient, who weighed 250 pounds and who had cardiac asthma, had a terrific cough whenever she lay on her back. She was kept reclining only three hours and obtained a good result.

Where glaucoma exists with cataract, external filtration can be established by removing a piece of sclera with the Holth punch underneath the flap (Figs. 4 and 5) before the lateral sutures are

closed. If this is done a water tight suture or additional sutures are placed.

Figures 6 and 7 are schematic illustrations of the action of the erisiphake on the cataractous lens.

The eyes are left undisturbed for forty-eight hours and then inspected and treated with bichloride ointment. Occasionally we inspect them in twenty-four to thirty-six hours. When they have been inspected for some source of discomfort, a few hours after the operation, we usually have found the anterior chamber reformed with a clear, black pupil. The unoperated eye is left uncovered after the third day. Patients can usually be up in a chair on the third day.

Where this conjunctival flap technic (Figs. 5 and 6) is used, patients can be propped up after a couple of hours, or next day at

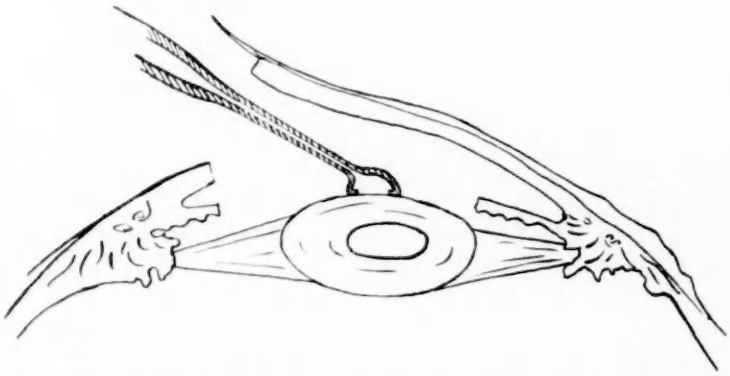

Fig. 6. (Schematic) Canula in anterior chamber placed evenly on lens capsule. Iridectomy above.

most. This is a distinct advantage in elderly people. They do not stand being bedridden and it is well to get them out as early as possible.

At the end of a week the operated eye can be left uncovered part of the time. The rapidity with which the eye clears is very noticeable. Redness and operative hyperemia usually are absent after a week or ten days. Some cases have cleared so rapidly that with two patients we have done tentative refractions on the seventh day, two on the eighth day and several on the tenth day, and many have been given glasses to wear on the fourteenth day. There is no lens substance or capsule to absorb or to set up a

secondary inflammation. Postoperative iritis has been conspicuous by its absence. Secondary infection is less likely to occur. There is no danger of a secondary membrane or cataract forming to obstruct vision.

COMMENT.

It is true that with this or any intracapsular method the average operator is more apt to have a bead of vitreous present or some loss of vitreous occur than with the capsulotomy method if he is content just to deliver the solid nucleus and leave the debris and capsule to absorb. If the surgeon attempts to remove

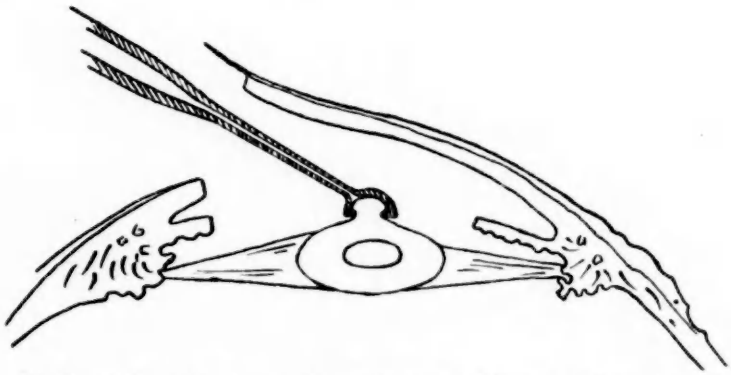

Fig. 7. (Schematic) Lens and capsule moulded into cup of canula and held as with forceps.

these either by irrigation or by forceps as most surgeons do, he is just as apt to have vitreous loss as in a careful intracapsular method. H. W. Woodruff[7] said in discussing the paper of Lloyd Mills, "It would have been better to have performed the Barraquer operation." The patient has a chance at something nearer the ideal at no additional risk. Arnold Knapp[8] has demonstrated conclusively that iritis, uveitis, and secondary glaucoma are caused by retained capsule and débris.

Colonel Elliot[9] advises that patients should not defer cataract operation because the other eye still has vision. His arguments are excellent and well taken.

The Barraquer-Green technique can be used in all uncomplicated types of cataract except where the lens is dislocated. We do not use it in young people. We prefer to do a discission and then remove it by the Teall method as described by Dean[10].

BRIEF ANALYSIS OF RESULTS.

Our patients are all private ones and we have been able to keep in touch with most of them. We are disappointed if we do not get vision of 20/30 or better in uncomplicated cataracts and 20/15 and J 1 is common. Mrs. S., the case I reported in my first article,[11] then 76 years old, now 83, still has vision of 20/15 and J. 1.

Our experience with phacoerisis covers a seven-year period in which over two hundred cases have been operated upon. A grouping of cases by hundreds follows:

	A	B	C	D	
	Presentation or loss of Vitreous	Capsule Intact	Retained Capsule Needling Necessary	Useful Vision not Obtained	Soft Eyes
1st Group	22%	72%	2%	1%	0
2nd Group	17%	81%	3%	4%	1
3rd Group	12%	85%	0	0	0

No globes lost.

The case in group one in which useful vision was not obtained had only light perception on nasal side of field before operation. The lens was removed intracapsularly and the eye found amblyopic except for light. This case had an unusually rapid convalescence, no vitreous loss and is a perfect looking eye. In no other instance where the cataract was removed intracapsularly has there been a failure of vision or a secondary inflammation of any kind. In some of our earlier cases considerable vitreous loss occurred. It had no influence on the visual results. Some of our finest results were in early cases where considerable vitreous was lost. Several elderly patients have died but they retained their vision to the end. Improvement in technic has reduced the vitreous loss.

In group two, one soft eye resulted. This case had had a history of previous pain and inflammation and had been blind for ten years or more. There was dim light perception, tension 40 (Gradle Schiøtz) at first examination. This was reduced by treatment and preliminary iridectomy. There were several synechiæ. The anesthetic was not sufficient, perhaps, and the patient became hysterical and uncontrollable. The lens was black. A large vitreous loss occurred.

The 5 per cent in group two, of eyes without useful vision, seems large, but when we consider that the cases were not selected and that fourteen cases in this group were highly com-

plicated or pathologic cataracts, it changes the complexion of the statistics. If one selected only uncomplicated cataract cases the percentage of visual failures would be greatly altered. Nine of the fourteen cases were operated on successfully and have good vision in spite of the complications. All of the fourteen cases had either a history of inflammation, pain indicating glaucoma, or synechiæ, indicating a previous iritis, or all three symptoms together. Five of them had increased tension.

Case two, under D, of group 2, had hypermature cataract and a history of previous pain and inflammation. Light perception was weak on temporal side of field. Pupil dilated poorly, necessitating small canula. Capsule was ruptured and a solid lens expressed by pressure. No vitreous presented. The capsule was retained. There was a good pupil and no needling was done. Secondary iritis followed. Convalescence was slow. Result was light perception and hand movements at two feet. Relief of symptoms.

Case three, under D, of group 2, gave history of general health poor, "neuralgia," discomfort about eyes and gradual loss of vision. Light perception. Tension 45 (Gradle Schiøtz). Shallow anterior chamber, poor incision, capsule ruptured, lens expressed, small retained capsule. Good pupil, no needling. Slow convalescence, no vitreous loss. Result: light perception. Relief of symptoms.

Case four, under D, of group 2. History of pain, inflammation. Light perception weak. Tension 32 (Gradle Schiøtz). Shallow anterior chamber. Capsule ruptured. One needling gave fair vision but this gradually failed. Glaucoma. Treatment not followed consistently.

Case five, under D, of group 2. Male, aged 47 years. No eye complications except myopia; very prominent globe. Wore sphere minus 3.00 combined with cylinder minus 2.50. Mature cortical diabetic cataract right eye. High blood sugar. Was exceedingly apprehensive and nervous and difficult to control. He had a marked contraction and spasm of right facial muscles when excited. A small preliminary iridectomy was performed three weeks previously and the blood sugar reduced. Patient was large and very muscular. The suspensory ligament was tough and the capsule ruptured as the lens delivered. The wound bled profusely and blood constantly filled the anterior chamber. It was difficult to perform any toilet on account of the patient's

extreme nervousness. The morphin and atropin given preliminary to operation had little effect or rendered him more excitable. The facial spasm started after the incision and continued for several hours. The eye bled continuously for three days so that dressings were saturated. After this had subsided, the iris was found to be prolapsed into the wound. A very stormy convalescence ensued, complicated by profound iridocyclitis and keratitis with diffuse corneal opacities. Potassium iodide and mercury were given in large doses for their alterative effect. Much improvement followed their use. A Wheeler incision through the iris was performed with a good pupil resulting, but the hazy cornea allowed only vision for moving objects at fifteen feet.

Three of the seven successful complicated cases in group two had glaucoma with the cataract. All three had preliminary iridectomy. The lens was removed intracapsularly and good vision resulted with convalescence only slightly longer than uncomplicated cases. In group three, five cases complicated by glaucoma have been operated on successfully.

In group three, one syphilitic case with cataract, a partly opaque cornea (superficial keratitis improved by treatment) and complete annular synechia was operated on with the capsule intact. The lens contents was entirely fluid. A preliminary iridectomy had been performed three months previously. The iris was released by sphinctertome just before the lens was extracted. The patient had seen practically nothing for ten years. Final vision was 20/40. The other eye has the same conditions but with only about three-fourths synechia and less corneal opacity. Operation will be attempted later.

Male, aged 69 years. Right eye crossed since childhood; cornea partly opaque (keratitis) with history of much inflammation, several synechiæ; was operated on successfully; vision 10/30. Left eye cataractous. (Since operated on. Vision 15/30.)

Male, aged 86 years. Right eye blind sixteen years; soft Morgagnian cataract removed intracapsularly with iridectomy just preceding the extraction. Counted fingers thirty-six hours after. Final vision was 20/20 and J. 1. Left eye also blind. (Since operated on with good result.)

I wish to state emphatically that in no instance has vitreous loss ever occurred because of the suction. When a bead presented or a drop or so was lost, it was as a result of the expres-

sion. It would have occurred with the combined forceps technic or any other intracapsular technic. It is always less apt to occur than with the Smith technic and if controlled as outlined, it need not be feared.

We have had three cases of secondary hernia of the iris occurring some time after the extraction. Two were treated successfully. The third case was one of known glaucoma and cataract and passed from our hands after three months with final outcome unknown. Vision was 15/40 when treatment was discontinued.

One case with a history of much previous inflammation on which an intracapsular extraction was performed had a thin hyaloid membrane. It did not interfere materially with vision.

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DISCUSSION.

DR. H. W. WOODRUFF, Joliet, Ill.: I first became interested in the suction method of grasping the lens for its extraction in capsule in 1910, at the time that Hulen published his paper on the subject. I invited him to come to Chicago to demonstrate his operation on cases which we would furnish him at the Infirmary, and to present a paper before the Chicago Ophthalmological Society. He did not accept the invitation. I then made an attempt at the operation, with the assistance of V. Mueller and Co., who made for me some cannulas with tips very similar to the Green-Mueller apparatus, and with an opening on the side so that the fingers would control the vacuum. We used the Beck-Mueller pump used in tonsil operations. I was not able to produce sufficient vacuum to grasp the lens and gave up the attempt.

After Barraquer brought out and demonstrated his method, and the Greens, in cooperation with Mr. Mueller, also produced a vacuum pump

and a simple cannula without valves, I again took it up, and so successfully that at the March meeting in 1926 of the Chicago Ophthalmological Society I showed seven eyes in five patients upon whom I had performed the operation. I followed the Barraquer operation as nearly as possible, using, however, the Green-Mueller apparatus. The small peripheral iridectomy and the delivery of the lower border of the lens first (the so-called tumbling method). At this time I had had fifteen cases, with failure of one, due to the lens falling back into the vitreous at the moment of its beginning exit through the wound. All the others were highly satisfactory, showing the small round and clear pupil with the peripheral small iridectomy. Ten more operations were performed with another lens lost in the vitreous, and one in which a fluid lens together with vitreous were drawn up into the instrument. This last case should not have been selected for the Barraquer method, as any of the traction methods would have delivered the lens without vitreous loss, I believe.

In giving publicity to a method which is such a radical departure from the older methods, the bad results and their reasons should also be brought out. The work of Barraquer and the careful technique, as well as the perfection of his instrument, are epoch-making in the history of the cataract operation. Note, however, that his followers in this country modify this and that detail in the procedure. The Green-Mueller vacuum pump is a marvel of the manufacturer's skill and it is certainly just as effective in producing the needed vacuum. How much advantage there may be in the respective erisiphakes one over the other, I am not certain. The Barraquer instrument gives one vacuum control with the finger on the valve, while the Green-Mueller electric current control is with the foot.

I know there are quite a number of these instruments in various parts of the country and I know some of them are successfully used, as Dr. Wolfe has demonstrated in his case. I think he is a man of courage, living in one of the smaller centers, having no access to a public clinic, but operating only upon private patients. Whether these eye surgeons whose work is in similar communities should follow his example is open to argument.

The subject of cataract is such an involved one and of such great importance that no one should dogmatically advise one just beginning to operate to follow this method; nor one who is reasonably successful with a certain technique to change to something else because he himself is an expert with it.

If it is in order to criticise Dr. Wolfe at all, I would suggest that in a given number of cases there would naturally be patients upon whom many conservative intracapsular operators would advise the capsulotomy method—those, for instance, with prominent eyes upon which only the safest operation should be selected; also those under sixty years of age. The Greens attach much importance to the wrinkling of the cornea horizontally after the incision is made. They consider this a contraindication to any intracapsular operation. This observation has not been noted by

other operators, but if there is a tendency for the wound to gap when there is no external pressure a capsulotomy should be performed.

As Dr. Wolfe states, operators are becoming more and more interested in the intracapsular operations and ophthalmology owes something to the pioneers in these various methods. There are, however, so many modifications, expert operators are constantly changing one detail for another, that the subject is by no means simplified. This has always been a debatable subject and probably will continue to be.

Dr. Wolfe makes a half-section of the cornea. Barraquer advises two-fifths of the cornea and an incision entirely in the corneoscleral margin. I would suggest that the incision might vary according to the age of the patient, inasmuch as the cornea grows smaller and the lens larger as age advances.

Dr. Wolfe advocates in some cases a conjunctival flap, prepared before the incision is made. I would recommend in place of this the regulation Van Lint sliding flap.

When Barraquer visited this country and demonstrated his operation, in not one case did he lose any vitreous or have iris prolapse. He had a few ruptured capsules; one dislocation of the lens into the vitreous. I believe one should be careful about making the operation too complex as to additional details. Simplify it as much as possible, consistent with safety.

I shall conclude with these observations: If you have the opportunity of operating on only a small number of cases, say twenty-five or less, in a year, operate on mature lenses with the combined capsulotomy method, preserving a normal pupil if possible. If you are in a large clinic, perfect yourself in some intracapsular method. There is certainly a place for it in ophthalmic surgery. It seems to me that in the old days, when we waited for lens maturity and even performed ripening operations, results were better than operating on immature cataracts by capsulotomy unless the lens was sclerosed. Cortical lens substance is certainly not well borne by old people. The uveitis induced may be of such long duration that even though the cortex is finally absorbed, corneal and vitreous opacities and glaucoma may seriously interfere with vision or cause the loss of the eye. This does not apply to young people, as is well known; otherwise the operation of discission of soft cataract would not be the operation of choice in such cases. Whatever part individual idiosyncrasy may play in the absorptive process in adults, age, I believe, is the more important factor.

The use of the forceps, as advocated by Knapp and others, is safe; but in my hands the capsule ruptures so frequently that it is the exception to succeed in removing the lens in its capsule.

Over the door of a building on South Dearborn street in this city there is this sentence: "The excellence of any art consists in the complete accomplishment of its purpose."

Dr. W. B. Lancaster, Boston: If anyone asked me about taking up the intracapsular operation I should give this advice: First, perfect yourself in the capsulotomy operation until you can operate on average cases

with loss of vitreous in decidedly less than 10 per cent, and iris complications decidedly less than 10 per cent. Then you may try the intracapsular operation with forceps, acquiring the ability to manipulate in the anterior chamber as is necessary in the intracapsular operation. After you have acquired a sufficiently good technique to extract in the capsule with forceps, then you may try, if you wish, the vacuum cup.

The deciding factor between these methods is probably the result, and it seems to me that some of the results published are misleading. For instance, Dr Wolfe said that in only a small percentage of cases did he succeed in extracting with forceps without tearing the capsule. And Dr. Knapp refers to the fact that while forceps extraction is the safest method it fails in a considerable percentage of cases because the capsule tears. Recently Safar reported 52 cases. His technique was almost exactly like the method I have used for a good many years, and his results, like mine, were so much better than those previously reported that I am convinced the method followed is largely responsible. The essential point in which Dr. Knapp's operation and mine differ is that Dr. Knapp advises you to grasp the capsule with the forceps and dislocate the lens, then lay aside the forceps, pick up the Daviel spoon and express the lens *a la* Smith. Török in the same way dislocates the lens with forceps and then expresses it with the spoon, but does not release his hold with the forceps. Elschnig and many others have followed this method.

In 1916, in discussing Török's paper, I advised that instead of following this plan of Knapp's, which Török also followed, of first dislocating the lens with the forceps and then expressing it, you apply some pressure *before* grasping the lens. Most capsules are easily grasped—some are difficult—but you will be able to grasp a larger percentage without doing any harm if there is a little counter pressure on the outside as you seize the capsule. Second, do not rely upon the forceps alone to dislocate the lens. The forceps plus the outside pressure will succeed in dislocating the lens without tearing the capsule in a much larger percentage of cases. Safar's figures are 92 per cent, and my own are considerably larger than those given where dislocation is done with the forceps alone. They are better than the results of vacuum cup operations. Not only is the average better, but the number of disastrous results is far smaller.

I ask you, therefore, to consider carefully the results of extraction in capsule with forceps when the best technique is followed, and compare them with the results of the vacuum cup method. If you find, as I think you will, that the results are better with the forceps, that should settle the matter. If, however, you should find some operator whose results with the vacuum cup were as good or better than ours with the forceps, then you should give consideration to these additional points: Which is easier? What of the complicated apparatus which may not be in good working order? Were the results obtained only after a large number of operations, perhaps thousands? Was the operator gifted with very exceptional skill?

Dr. S. G. Higgins, Milwaukee: Dr. Wolfe has found sutures helpful. In this connection I wish to refer to a suture especially suitable for the

Barraquer operation, both for closure of the wound and also for the purpose of raising the cornea to prevent operating in an open field. For purposes of cutting off presenting vitreous I am partial to a cornea-conjunctival suture. The suture is placed as a part of the preparation before the incision is made. Had I seen Ziegard's article before coming to this meeting I would call the suture I use a modified Ziegard suture. Ziegard's is in turn a modified Kalt suture. Neither of them mention the use of their suture for raising the cornea. They both wished to close the wound at the limbus. My idea was to set the crown-like cap of the cornea on the cut sclera. Kalt's suture allows one-half millimeter for passing of the knife; Ziegard permits one millimeter. By inserting the thread at greater distance from the limbus the knife may emerge where the operator chooses, which is a consideration in the Barraquer operation.

DR. A. S. GREEN, San Francisco: During the last four or five years especially, the intracapsular operation has become increasingly popular. I fear that Dr. Wolfe's excellent paper today will inspire many more to undertake the operation, and in view of this I desire to give a few notes of warning to save the intracapsular operation from unwarranted condemnation and probably the loss of many eyes.

Early in our cataract career we, like Dr. Wolfe and others, thought that intracapsular operation was suitable for practically all forms of senile cataract. But before long, and at the expense perhaps of some avoidable losses, we began to realize that we were overenthusiastic. We then began more carefully to select our cases, so that in the past twelve years or more our intracapsular operations have run consistently about 65 per cent, with the consequence that our general results have been much better.

First of all, we feel that Dr. Wolfe's lower age limit is too low. One of his cases is 47 years of age. We eliminate practically all cases for intracapsular operation under the age of 55, and some cases under the age of 60. But there is no upper age limit; the older the patient the more suited for the intracapsular operation.

We do not agree with him that the technique can be used in all uncomplicated cases, but that it can be used to better advantage than the capsulotomy operation in some complicated cases. For instance, in old iritis or iridocyclitis and diabetes, we prefer to do the intracapsular to avoid precipitating inflammation afterward. On the other hand, besides the restriction due to age, we also eliminate myopic eyes, prominent eyes and those with glaucoma. Of the remainder the final decision as to whether we will do an intracapsular or capsulotomy operation is only reached after the section is made, no matter what the tentative plan of operation had been beforehand. If, after the section, there is even the slightest gaping of the wound, or if horizontal streaks appear on the cornea from the point of entrance to the point of exit, then under no circumstances must an intracapsular operation be undertaken. If, on the contrary, there are vertical depressions or a dimpling or dropping down of the cornea, then an intracapsular operation may be performed without risk.

Since adhering to these very positive indications and contraindications we have reduced our vitreous losses to less than 3 per cent and ruptured capsules to less than 2 per cent.

Dr. W. A. Fisher, Chicago: The essayist states that he requires five specially skilled assistants. Smith stressed the importance of the highly trained assistant so strongly that this one point prevented many from even attempting his operation. Smith, Barraquer, Elschnig, Villa Coro, Poyales, Holland, Cruickshank, Knapp, Vail, Verhoeff, King, Millette, Green, Pratt, Suker, Gradle, McReynolds, Benedict, Nugent, Castroviejo and very many others have only one assistant whose only duty is to hold the lids. If the operator and the nurse who holds the lids are qualified, all will usually proceed safely, whether the capsulotomy or any of the intracapsular operations are used. One of my associates, Dr. Nugent, operates by the Barraquer method, but he has never seen Barraquer operate. Another associate, Dr. Ramon Castroviejo, operates with the Verhoeff forceps, and he has never seen Verhoeff or Kalt operate. I, personally, prefer not to decide which intracapsular method to use until after the incision has been made.

If blood in the anterior chamber obstructs the field of operation and cannot be removed, or if the pupil is too small, the Smith method is selected, but with many modifications. These modifications seem imperative to me, but might not appear necessary to Smith and others. (Ill. Med. Jour., Sept., 1929.)

There are more operations performed by the Smith method than by all the other intracapsular methods combined, and this may be due partly to the fact that the Smith operation does not require such perfect ambidexterity as the Barraquer or forceps operation. Both the Barraquer and the forceps operation, except that of Knapp, require a pull upon the capsule with one hand while pressure is made with the other. In the Smith-Knapp and the capsulotomy method the extraction is done entirely with one hand, which makes these techniques less difficult to acquire than the Barraquer or the forceps.

Congratulations are due Dr. Wolfe. We all know he would not do two hundred Barraquer operations unless he had obtained good results from this method, because he must get his operations from satisfied patients, but why make the technique so complicated?

Elschnig reports many intracapsular converts in Germany, and two intracapsular papers by the Elschnig method have recently come from Vienna. The testimony from all over the world seems to be in favor of some form of intracapsular operation. Many operators believe the time is near when some form of intracapsular operation will be generally adopted, due in part to better methods of anesthesia.

Dr. O. B. Nugent, Chicago: In my hands the Barraquer operation is the ideal method of cataract operation in that it combines into one system the best attributes of all methods thus far devised. Results by this method should show no more burst capsules than with the Smith operation, and much less than with the forceps method. The percentage of vitreous loss should be the same as with the forceps method.

No cataract method can justly be classed as being simple or easy for anyone to perform. It becomes easier and more simple as one gains experience.

The method of combining expression and traction described by the essayist I have been using for some years. I first began to use it while operating in India, four years ago, and found it so useful that I have devised a forceps which serves in three capacities: first, to pick up the cornea and conjunctival flap to facilitate the application of the erisiphake to the lens; second, for expression, as it resembles the Smith hook when closed; third, for tying the sutures. It serves to simplify the operation and saves time as well.

Our method further differs from that described by the essayist in that we prefer to make a small peripheral iridectomy after the lens is delivered. This is one of the most difficult steps in the operation, and we have been able to simplify it by using a small iridectomy hook which facilitates the picking up of the iris with greater ease than with the iris forceps as was formerly done.

I wish to emphasize the point Dr. Wolfe mentioned, that vitreous loss is only encountered after the lens is extracted and not during its extraction, and to stress the necessity of great care while performing the toilet.

Dr. R. Castroviejo, Chicago: It has been very well proven that the intracapsular extraction has many advantages over the extracapsular. It gives better vision, less postoperative reaction and with modern technique, statistics have shown that the loss of vitreous is not greater by the Barraquer or forceps intracapsular method than by the extracapsular.

Concerning the limit of age of 60 years for the selection of cataract, as Dr. Green stated in his discussion, we do not agree with him because, as cataract is a pathological process, the sclerosis of the zonula fibers does not always progress with age, similarly. We might find lenses in very young patients very easily extracted intracapsularly, and some in old patients very difficult of extraction, which shows that the sclerosis of the zonular fibers cannot be diagnosed before the operation by any method, and only the attempt to remove it gives us the knowledge of the resistance of the zonula fibers.

We have to agree with the essayist and with the doctors who discussed his paper that the Barraquer operation gives less rupture of the capsule than the forceps, but the number of intracapsular operations with forceps can be increased by the use of proper forceps, for instance, the Verhoeff's forceps, which has several advantages over the others, as the tongs have flat surfaces which do not cut the capsule, and another mechanism which does not allow excessive opening of the forceps and the subsequent grasping of too much capsule; then the vertical movement of the tongs is eliminated, and, with that, another chance of rupturing the capsule.

About the question of iridectomy, it might be considered as a complication, and the danger of traumatism of the eye is as little as possible, and most of the thickening of the hyaloid membrane after cataract operation is found in cases with iridectomy.

Dr. Otis R. Wolfe, closing: Dr. Woodruff spoke of losing the lens in the vitreous, and I have had the same experience. I delivered it with the loupe and the case had no complications and a very good result, but it taught me a lesson—that I was using too much vacuum. It is important for the man who wishes to use this technique, if he is not sure of the strength of the capsule or the sclerosis of the zonule, to use a lower vacuum rather than higher, and it is surprising how sometimes you can deliver the lens with very low vacuum. A point that should be stressed is that it is absolutely necessary that one be familiar with other techniques and be prepared to meet complications that come up by delivering the lens in any external capsule operation.

Dr. Green misunderstood me about the advisability of a simple intracapsular operation in complicated cases. I think it is the operation of choice in those cases.

One thing that I consider particularly important in this operation is that the stitch is tied, not only looped but tied, and my associate draws the sutures tight, and if the vitreous bulges we immediately close the eye and wait ten minutes. That has probably been the most valuable thing in the whole procedure to prevent vitreous loss. It is surprising how after ten minutes the vitreous will settle back and you have completely obviated loss of vitreous.

Analyzing my results, I will first state that the cases were not selected. Improvement in technique is gradually but positively decreasing the number of cases in which the capsule is ruptured or vitreous lost. Our limited experience indicates that while loss of vitreous is highly undesirable, even in small amounts, it is less to be feared than the complications resulting from retained capsule and lens débris.

I do not wish to convey the impression that the method is easy or without objections. It is conceded to be a difficult technique, requiring delicate manipulation of complicated mechanical apparatus. The arguments in its favor are those for any intracapsular operation, plus the fact that it is less liable to tear the capsule in certain types of cataract, especially the immature. In the last analysis it is a question of end results.

HERPES AND ALLIED CONDITIONS.

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"There is scarcely any cutaneous disease which may not be seen occasionally on the eyelids. Herpes I have often met with, both in children and adults. It runs its usual course of about a fortnight, leaving pits, like those of smallpox. Not infrequently it attacks the cornea, a vesicle having its seat there, ending in an ulcer." Mackenzie. A Practical Treatise on the Diseases of the Eye, 4th Edition, 1854, p. 149.

Hutchinson seems to have been the first to describe herpes zoster ophthalmicus in detail. He states that Rayer had reported a single case, that the Danielssen & Beck Atlas of Skin Diseases contained an excellent portrait of a case and that there was a very good wax model in the Guy's Hospital collection. In addition, the quotation from Mackenzie, which opens this article, completed the list of available material on the subject, in Hutchinson's time.

It seems strange that this clearcut clinical entity, with such serious visual curtailment in sequence, failed to enlist the interest of an earlier clinician, for it certainly never went unnoted. Hutchinson believed the eye escaped unless the cutaneous branch of the nerve supplying the side of the nose was involved. He reported three groups of cases to support this idea, but the rest of his observations have been amply confirmed. He noted the pain in the cutaneous area involved, even before the eruption appeared; the corneal ulceration with visual loss due to the scar and the appearance of the corneal ulceration after the skin eruption, not before or with it. Horner called attention to an increase of temperature of the skin and an increased sweating of the affected skin area with reduced intraocular tension. Wyss was the first to examine the Gasserian ganglion and found round cell infiltration, small hemorrhages and degeneration of the nerve fibers running to the first division of the fifth nerve. A few years later Sattler confirmed these observations, which ought to convince everyone that measures directed toward relief of symptoms caused by malfunction of the end organs connected with diseased ganglion cells should be supplemented by treatment directed to-

ward the cause or the site of the primary lesion. From time to time cases complicated by optic nerve atrophy, paralysis of part or all of the branches of the third nerve, permanent or prolonged loss of sensation of the affected cornea, enlarged pupil with sluggish reaction and other trophic effects have been added to the literature.

In contrast with the usually severe herpes zoster ophthalmicus is the milder corneal ulceration without skin eruption bearing the title of herpes cornea febrilis. It occurs after antityphoid injections, vaccination, therapeutic inoculations of malaria, salvarsan injections, pneumonia, malaria, cerebrospinal meningitis and even with common colds and herpes of the upper lip or nose.

The blister of this disease type contains a virus that can transmit the disease to the rabbit cornea, while the zoster blister does not do this. The eye symptoms of herpes zoster ophthalmicus are secondary to the virus acting upon the Gasserian ganglion; trophic changes with secondary infections, while the febrile type is the result of direct action of a specific virus upon the end organs and other tissues of the cornea, with secondary infections, an important factor. While the febrile type may be just as severe in its effects upon the cornea, iris and vision as the zoster it is usually milder and may leave very light traces behind.

Both of these forms of herpes are rare as compared to keratitis dendritica and its various forms. This is an eruption of a group of very fine blisters that lose their epithelial covering and link up to form a linear ulcer resembling the small branch of a tree. Corneal sensitivity is lowered or lost, presenting a picture like zoster on a small scale. It may begin very suddenly with a sharp sting or a burning sensation that makes the patient think something has blown into the eye. While it may develop without any indication of other disease or definite cause, it often follows grippe and grippe colds. Inoculations of the human cornea from lesions produced on rabbit corneas have produced typical dendrites, and scrapings from this type on the human cornea have given the same results as the herpetic blister. Since the type of grippe that appeared here first in 1918 has held sway, there seem to be many more dendritic ulcers than before that date.

Because of the blister formation, despite the larger size of the lesions, keratitis bullosa soon came into this grouping (with all secondary types such as are found in glaucoma or degenerative conditions of the eye and the type associated with constitutional

diseases, with blisters on the skin, placed by themselves in a quite different class). The painful corneal lesions characterized by epithelial shreds, known as filamentous keratitis, were considered an accidental group only, but not distinct from the bullous forms.

Animal experimentation and exfoliation after apparent healing of traumatic corneal ulcer strongly suggested that this condition likewise belonged to the herpes group. It is believed that trauma permits the penetration of a virus previously present in the subconjunctival sac, which in turn causes the local pain (often severe and out of all proportion to the apparent lesion), and the later blister formation and loss of corneal epithelia. Italian observers have investigated this condition and believe that there are those who might be classed as "herpes virus carriers." The virus is inert until it passes the epithelial barrier. No one has been able to isolate a germ or other specific agent, because stains show nothing and the virus will pass through a filter. If fluid from an herpetic vesicle is introduced into certain animals (such as guinea pig, rabbit or white mouse but not the dog or white rat), the corneal lesions appear, irrespective of the manner or site of introduction. A type of encephalitis also develops, and if the virus is introduced in one conjunctival sac the second eye is affected much after the way sympathetic ophthalmia happens. Some investigators have also been able to produce lesions much like impetigo contagiosa. This herpetic virus can be passed through cultures or animals and remain true to type, but certain samples seem to affect the nerve side of the organism more than the cutaneous, while others have an affinity for the skin. These experiments, the frequency of herpetic lesions after grippe and the occurrence of postgrippe lethargic encephalitis are the basis of the belief that the virus of herpes is the agent to be blamed for lethargic encephalitis and the other infectious nerve ailments. This latter group includes multiple sclerosis, acute ascending paralysis, poliomyelitis and poliomyelitis superior. Herpes seems to be an infectious disease because it is generally accepted that an attack of shingles protects against other attacks, and the relapses of herpetic lesions of the cornea can be explained on entirely different grounds, which will be discussed later. Clinical experience with cases beginning as dendritic keratitis or herpes corneæ febrilis, and which later pass into what conforms to every requirement for a diagnosis of disciform keratitis, inclines us to the view that this is also of the herpetic group. Either this is a new clinical entity or disciform

keratitis is at times featured by ulceration and a loss of corneal sensation. Many observers in Europe now class this unique condition as the result of local injury, allowing entrance of the virus to the deeper tissues of the eye, whence it may penetrate the Gasserian ganglion and other cerebral tissues by traveling along the nerve sheaths or even along the vessels. The slit lamp gives much valuable information in connection with this large group of corneal lesions and teaches the importance of regarding every eye afflicted in this way as unable to protect itself against invading organisms or the evil effects of exposure to air and dust. Practically every case of the group has a lowered corneal sensitivity and very evidently a lowered vitality that requires constant closure and covering of the eye, both indoors and out. If the initial lesion should heal, for some time the cornea is below par and until the normal sensation has returned should be protected and a local antiseptic used to keep the conjunctival sac free from dangerous agents. If we were allowed to select but a single method of treatment for herpetic cases, my selection would be the constant covering of the closed eye. Next to this measure is the use of radiant dry heat to the open eye, and the most satisfactory form for the usual case is the glowing heat given off by the so-called infra-red lamp. This lamp is also known as the thermolite and has a large carbon spiral that never gets hotter than the cherry red stage. It should be placed at 20 inches from the patient, with the rest of the face and forehead protected by thick towels, and the affected eye directed steadily at the glowing spiral for 15 minutes at a time. These seances can be repeated four times a day. In some cases, especially the severe types of ophthalmic herpes, the Shahan thermophore is the best treatment for ulcers that refuse to respond to other methods of treatment. The glowing heat from the lamps mentioned above is said to be only the infra-red rays, but no proof of this has come to my attention. The best local antiseptic seems to be optochin in a one-half or one per cent solution. This is entirely a clinical demonstration, and no experiments in the laboratory have been made to confirm or deny, but the results are certainly more pleasing with this than with mercurochrome or argyrol. The pain of herpes zoster in the eye is not usually severe unless the deeper structures are invaded, but pain in the temporal region is often agonizing. It is usually much worse at night, and the only thing that reaches the pain of the severe herpes zoster ophthalmicus is an applica-

tion of the X-ray. The dosage must be rather heavy but not more than a single application is necessary to produce marked benefit. A second or third application may be required later. It seems that an increase of intraocular tension is regular in these severe cases and the X-ray has a most favorable influence upon the tension also. In the section given to case reports these features will be noted. If corneal ulcers are present, it is wiser to omit the tonometer and depend upon the fingers, even if we expose ourselves to the criticism that the digital method is inaccurate and unscientific. The alternative is the knowledge that we have done harm to a cornea lacking vitality, which is, of course, no concern of the critic.

The after-effects of severe herpes are indeed severe. The most usual damage is the corneal scarring, both deep and wide. The slit-lamp, during the acute stage, shows a fish skin cornea with flakes of opacity throughout the deep corneal layers, and deposits upon the posterior surface of the cornea indicating ciliary involvement. Synechiæ are frequent and permanent. In some cases the iris is eventually decidedly thinned and the color changed to an extent deserving the name of heterochromia. The pupil is rather regularly larger than its fellow, and the more severe the case the greater the disparity and the longer its tenure. What happens in the interior of the eye is usually concealed from view by the corneal opacity or the deposits in the pupillary area. Optic nerve atrophy and paralysis of some of the muscles supplied by the third nerve happen now and then. The older the patient the more severe the case, and it is not so unusual to find a severe case of herpes, suffering for a year or so, with severe headaches at night and a general debility that is out of all proportion to the visible signs. The danger of drug addiction is added to the other unpleasant features of these cases, but the number of deaths, even among the older patients, is fortunately small. Herpetic lesions of the eye are much more numerous than is generally conceded, and the advice of Theobald to employ a little wisp of cotton to pick out the case with lowered corneal sensation has been neglected. Nor has the rôle of corneal hypaesthesia and the associated loss of corneal vitality been appreciated. The average case of dendritic keratitis will heal under protection of the eye and the use of a local antiseptic. As soon as the patient is relieved he insists upon exposing the eye, which cannot withstand the drying effect of exposure to the air nor the hazards of city air with myriads of

minute dust specks everywhere. A relapse follows or the case may pass into disciform keratitis. This is not theory but the common effect of willful disobedience by the cocksure patient who is always right as long as he has no pain. It seems that fluorescein is rather generally used to detect the minute ulcer of whatever kind, but it is never so useful as in showing up the minute ulcer of dendritic keratitis in the very early stage. If this same dye is used in the earlier days of disciform keratitis minute breaks in the surface epithelia can be easily demonstrated, if several minutes pass between the instillation and the observation. The slit-lamp is especially useful in showing the minute defects and the final diffuse staining after the dye has an opportunity to soak in. Retroillumination will set off the minute ulcers to advantage, but direct illumination shows the staining better.

One of the symptoms recorded occasionally during the course of herpes ophthalmicus is increased intraocular pressure. Some observers seem to think that this increase of pressure within the globe accounts for the pain, and operations have been done to relieve the tension. While the number of the cases with increased tension coming under my observation is not great, the relatively high percentage of increased tensions has led me to believe that we ought to expect this tension rise in any severe case of herpetic keratitis (herpes zoster ophthalmicus and herpes corneæ), and also in severe keratitis dendritica and its later stages that take on the aspect of disciform keratitis. As has been mentioned, the X-ray has relieved both the tension rise and the pain that evidently originates in the inflamed Gasserian ganglion. As mild doses of the X-ray stimulate tissues but heavy doses inhibit or destroy, the effect upon the ganglion might be understood. The rise of tension may be explained upon the ground of hypersecretion of the ciliary body or stimulation of the sympathetic nerve. Sgalitzer experimented with dogs and found that the X-ray would inhibit the secretion of the choroid plexuses of the cerebral ventricles and came to believe that in this way was relief obtained in brain tumor cases. He had one striking case of escape of cerebrospinal fluid from the nose, with a diagnosis of probable pituitary disease, promptly relieved by X-ray therapy. The inhibition of cyclitic secretion by the ray would not seem unreasonable.

Of all the herpetic group none is more rare than keratitis punctata superficialis. Of this condition but two or three cases have been seen in twenty years. The classic features of this disease

are the minute superficial erosions scattered here and there over the cornea, their tendency to remain for some time, the total absence of any deeper or more severe sequelæ and the association with bronchial conditions. A type of subepithelial elevation covered by sound epithelia which permits no staining is seen now and then but much more frequent than the classic type described above. The cornea loses its acuteness, and if protected, seems to recover slowly and very little is left behind to show that at one time the cornea was liberally sprinkled with these minute elevations that are only semiopaque and never at all dense. Nothing has been discovered as to cause nor a treatment superior to the customary protection and local antiseptic. Gradle has described a keratitis punctata subepithelialis, which is a good name for this type, but whether it is really of this herpetic group is not yet settled. The punctata keratitis first mentioned seems to be a member of the herpes clan because lesions of its type have been experimentally produced in animals.

The conclusions that seem to flow from the discussion might be epitomized as follows: Herpetic lesions of the cornea seem to be quite common, and especially after grippe and grippe colds. An outstanding symptom quite characteristic of all of these lesions, including the traumatic case, is the lowered sensation of the cornea, which is easily detected by drawing a wisp of cotton across the corneas of the two eyes for comparison. With this lowered sensation is associated a lowered trophic influence, permitting the entrance of a virus that rapidly reaches the deeper parts of the eye, leaving a permanently damaged cornea. The corneal sensation is slow to return, and in severer cases remains permanently below normal. The so-called relapses are the results of damage to the surface epithelia by dirt or exposure to air currents which a normal cornea is well equipped to withstand but which is too severe a test for a cornea with lowered vitality. Glaucoma of the lesser degrees is often found with the severer cases of this group but rarely requires an operation, as both the tension and the pain coevil therewith can be controlled by moderately heavy X-ray therapy. If these patients are told why and how the relapses occur and supplied with a local antiseptic and the proper eye covering, further trouble can be practically eliminated, but there are some who can learn by severe experience only. The period during which these relapses can occur is certainly as long as three years and probably more. The slit-lamp will distinguish

the dendritic ulcer, even in the early stages, when to the naked eye or the loupe it is without definite characteristics. The blister is the characteristic sign of herpes and may be minute or large, but trophic disturbances and lowered corneal sensation are of equal or greater value in establishing the diagnosis because they persist after the blister has healed.

A type of profound keratitis with all of the appearances of disciform keratitis belongs in this group and has been under ob-

Fig. 1. Slit-lamp picture of fish-skin surface of the cornea in herpetic keratitis. The dark area at the top of the picture is the pupil area indistinctly seen because it is out of focus.

servation from a minute initial dendritic ulcer to the final stages, and also without the initial ulcer. The density of the cloud is usually the result of continued exposure of the eye through ignorance or disobedience, after the corneal opacity has formed. The initial crop of blisters in dendritic keratitis seems to appear with such suddenness that many patients are sure there is something

in the eye and seek its removal. So small may this group of blisters be that it is easily passed over unless a stain is instilled or the slit-lamp used. Laboratory experiments strongly suggest the virus of herpes as the cause of both dermatropic and neurotropic entities, including such nerve diseases as poliomyelitis, polioencephalitis superior, multiple sclerosis and acute myelitis. Both clinical and laboratory experience agree that among the dermatropic effects of this virus should be included not only herpes zoster ophthalmicus and herpes corneæ febrilis but also dendritic keratitis and its various forms, disciform keratitis or a type of keratitis profunda like it, keratitis bullosa, vesiculosa and filamentosa (when not caused by degenerative eye disease like glaucoma absolutum, etc., nor by a profound systemic disease), keratitis punctata superficialis and finally the relapsing traumatic ulcer. If we can influence our patients to protect the affected eye for a few days after the removal of a foreign body, and also whenever the corneal sensation is below par, the relapsing traumatic ulcer and the more serious corneal opacities that inevitably follow invasion of the deeper corneal layers can be avoided or minimized. Genuine ophthalmic herpes always produces such dense corneal opacities that the eye is useless for more than detection of large objects or motion. With the exception of relapsing traumatic ulcer, corneal herpes leaves a crippled eye which in addition to the corneal opacity has a lowered corneal sensation and vitality. In many cases this never returns to normal, and in most cases there are periods of higher and lower resistance, which must be taken into account if the patient is to avoid further trouble.

CASE REPORTS TO ILLUSTRATE THE CLINICAL ASPECTS OF HERPES AND ALLIED CONDITIONS.

Case 1. Severe ophthalmic herpes zoster, with increase of tension and severe pain relieved by X-Ray therapy. Relapse of ulceration occasioned by premature exposure; permanent loss of sensation of cornea. Mrs. M. K., aged 60 years, twelve weeks ago had an eruption of herpes in the hair area of the right frontoparietal region and six weeks later the right eye became involved. When she was seen first the cornea was clouded by a large scar with much thickening of the deeper layers. Numerous anterior synechiæ and the corneal sensation much reduced. Eye is not painful now but there is severe pain in the temple at night. Very moderate ciliary injection. Tension recorded by Schiötz instrument was 35 and 18 mm. respectively. The patient complained

bitterly of the pain at night and seemed very much depressed. Pilocarpin, eserin and cocain solution was instilled in the eye to relieve the tension, moist heat applied and mercurochrome 1 per cent solution instilled. If fluorescein was instilled in the conjunctival sac, and a few minutes allowed to permit the dye to penetrate, minute defects in the surface epithelia became evident. Ten days passed without relief of pain or the mental depression, when Dr. W. H. Dieffenbach gave one deep X-Ray treatment with immediate relief of pain. The tension was reduced to 21.5 and 15 mm. respectively. One month later the eye was white, the cornea insensitive and the tension 15-18 and 18 mm. respectively. The pupil was larger than its fellow and immovable. The adhesions could not be seen because of the cloudy cornea with much pigmented deposit upon its posterior surface. A fish skin surface was seen when the slit-lamp was used. Twice after this observation the patient insisted on going about without the shell over the eye and at once small ulcers developed. Two years later the patient returned for observation and was quite comfortable. There was no pain or redness, the corneal scar was thicker than ever and the surface much less sensitive than the other. For visual purposes the eye was useless.

Case 2. Case began with "something that flew into the eye." Disciform keratitis (or keratitis profunda) with recurring dendritic ulcers, severe pain in temple and moderate elevation of tension relieved by X-Ray therapy.

About three weeks before his first visit the right eye suddenly began to sting and burn as if something had blown into it, but the physician visited by him, could find nothing. Immediately thereafter the vision was blurred, the eye moderately red and painful. When he came here the eye was red, the cornea hypaesthetic, no apparent ulceration but minute defects visible after fluorescein instillation. The slit-lamp showed the typical fish skin surface with diffuse specks scattered throughout the cornea and some large clumps on the posterior corneal surface. The presence of adhesions suggested the use of atropin, but in about two weeks this was stopped because of increased tension, according to the Schiötz instrument, as follows: 25-30 and 15-18 mm. respectively, for the right and left eyes. The pain in the side of the head at night became very severe. The patient could not be persuaded to wear an eye patch and in a few days an ulcer of the cornea appeared internal to the pupil and typically dendritic.

In three or four days this had healed, but the patient did not wear the patch any longer than the eye pained and relapses occurred. A single X-Ray treatment relieved the pain and the finger tension indicated satisfactory progress in that respect, but because of corneal ulceration and the difficulty in healing no tonometer was used. From this point on the case was featured by repeated ulcerations, slow healing and relapses. He finally was in a bad way because of a large superficial ulcer that would not heal. There was little pain, the corneal sufface was insensitive, but nothing influenced the corneal erosion. He was now in other hands as I was out of town for the usual summer vacation. Dr. James Andrew saw him in consultation and promptly closed the case by using the Shahan thermophore. One year later the corneal sensitivity was seemingly as acute as the other but the eye was of little use because of dense corneal opacity.

Case 3. After-effects of herpes corneæ resembling heterochromia. Mrs. A. M., aged 58 years. During past year the vision of the left eye has been failing and is now counting of fingers only, mainly because of corneal scars with diffuse clouding. Moderate redness, no evident ulceration but fluorescein shows many fine defects. This cornea is hypaesthetic, surface is typically fish skin and there are "bubbles" here and there near the surface and many pigmented clumps on the posterior corneal surface. There are many synechiæ and the lens is decidedly cloudy. She had been to physicians for the original attack of "ulcer" of the cornea, but thereafter had attended to the eye herself. The eye was protected and local antiseptic used. This restored it to its normal whiteness but without improvement in vision. During the succeeding year the iris color faded and the slit-lamp showed very plainly the white radiating lines containing the blood vessels, not covered by pigmented tissue, as is the normal state. She must now and then cover the eye even indoors to avoid the irritation and redness and minute defects that develop otherwise.

Case 4. Dendritic keratitis developing during an attack of antral disease. Mrs. M. L., aged 42 years. Three weeks ago the eyes bothered her a great deal. Both were somewhat red, vision blurred and both lower lids puffy. The left antrum contained pus and it was later shown that there was a pansinusitis, but only the left antrum contained pus. Soon after the operation to drain the affected antrum a typical dendritic ulcer appeared on the left cornea, preceded by severe local pain and lachryma-

tion. This ulcer healed promptly under protection, moist heat and local antiseptic. The case is quoted as an example of the disease occurring during nasal accessory disease, of which type the literature contains a number of examples.

Case 5. Relapsing traumatic ulcer of unusual severity. E. C., aged 21 years. Struck left cornea with whisk broom last night. At first visit has a large superficial ulcer of the left cornea with much photophobia and lachrymation, and a small pupil. The other eye sympathizes so he had difficulty in getting about. The ulcer was cauterized with pure carbolic, argyrol instilled and local heat employed, but it was two weeks before the ulcer healed, and three weeks more before the eye could be exposed to light and air with comfort. All was well for four months when the exfoliation recurred over the entire lower half of the cornea, after it was struck by the corner of a theatre program. The entire raw surface was brushed with pure carbolic, the eye covered, atropin instilled, with relief of irritation in four days.

The relapsing ulcer history usually includes either a long period between the accident and the employment of protection to the eye or other treatment or the subject striking the eye with something which, from the surgical point of view, is decidedly unclean. The finger nail is the most common cause, but in this case the original wound was inflicted by a very unclean object, a whisk broom. The only feature of the second ulcer out of keeping with the conditions was the size of the exfoliation.

Case 6. Dendritic keratitis with every appearance of disciform keratitis with recurrence several years after initial lesion. A. E., aged 35 years, said something flew into eye four days ago. The eye was painful, moderately red, teared profusely and there was an ulcer at 5 o'clock with much surrounding corneal clouding. The slit-lamp showed this clouding to be a number of striæ in the deeper corneal layers, running in all directions from the lesion site, and the surface was typically fish skin. The eye had not been protected at all up to this time. This ulcer healed promptly and the corneal clouding cleared, leaving but a small patch of clouded cornea over which the fish skin surface was plainly seen. There was also a lowered sensitivity of this cornea. Three years later he reappeared with the history of something in eye a month before. After this had been removed the eye was more or less irritable and when he was examined the characteristic fish skin surface, with vacuoles here and there, could be found with the slit-lamp. There was a large disc of opacity

in the cornea apparently within and not on the surface. Fluorescein stained the deeper layers only after some minutes, but no actual point of entry could be made out. The lesion was below the pupil area so there was no effect upon the vision. Three months after healing of the second lesion the pupil was much larger than the other and the cornea was still less sensitive. The illustration shows the affected area of this cornea, one which is typical of the corneal surface after dendritic and herpetic keratitis. In some cases the individual flat elevations are very small, but in other cases they may be mixed with the larger spots as in this case.

RESUME OF LABORATORY EXPERIMENTS WITH HERPES VIRUS.

Gruter seems to have first shown that the contents of a herpes febrilis blister could reproduce its characteristic lesions upon the cornea of the rabbit. He did this in 1912, and in a year or so Kraupa, with his advice, was also experimenting along these lines. Lowenstein made his results public in 1920, and since then a large number of investigators have been interested in the ever widening aspects of a problem that seemed unimportant, but now are so inclusive that each group or investigator can only handle a part of the work. The technic employed began with cleansing of the surface of a blister. This was then carefully opened and the contents carried on a narrow knife to 5 cc. of normal salt solution. This made the standard solution from the upper layers of which some fluid was carried on a knife and two parallel lines made on the cornea to be tested. The contents of a zoster blister, a burn blister, eczema pustule, and pemphigus gave negative results. But the virus from the febrile blister gave consistently characteristic results. The wound is without irritation for 16-24 hours; at 36 hours there is strong ciliary injection and photophobia with considerable secretion in the conjunctival sac. Many fine blisters may be seen along the edges of the wound. These increase in number at right angles to the wound, often duplicating the tree like branches seen in practice. By this time the cornea is completely anesthetic. The disease reaches the climax in 6-8 days.

The disease may be carried from one rabbit to another and Gruter did this no less than 23 times without failure. The virus is easily killed, one-half hour at the temperature of 56° (132° F.) destroys it completely, but the solution above described was diluted 200 times and was still effective. If a cornea recovers

from one infection a sort of local immunity is established, which for some time protects against reinfection. It is generally believed by laboratory experimentors that the virus will pass through a filter, but there is no general agreement on the point. A double coccus is found in the blister contents, but its significance is in doubt.

Microscopic examinations of rabbit corneas at different stages are illuminating and show a loss of superficial epithelium as the first effect. There seems to be an accumulation of fluid in the upper layers of epithelium with very many very fine granules that are found nowhere else. There is a strong tendency to the formation of vacuoles in the cells with deformed nuclei still to be seen. Stargardt calls this the "balloonizing degeneration." Blisters are formed thus and the upper epithelia stripped off, but until the parenchyma is bared, it remains in good form. When a considerable area has been stripped, Bowman's membrane melts, the exposed parenchyma splits up, the surface parts becoming glassy and staining badly. The parenchyma away from this process is infiltrated with polynuclear eosinophilic leucocytes. Despite the lost tissue the affected cornea is much swollen and occupies more space than the original area. Descemet's membrane and the endothelial lining remain unaffected and there is no increase in fixed tissue cells. This pathology is in harmony with slit-lamp and direct observation.

Stocker instilled the virus in the conjunctival sac of the rabbit, which merely prolonged the incubation period, but did not alter the effects. Luger and Lauda successfully infected the human cornea and A. Fuchs and Lauda repeated the experiment. Levaditi, Harvier, Nicolau and Doerr found a virus in the human saliva that seemed to be closely connected with the epithelia therein. The local immunity gained by recovery from an infection is not limited to the eye operated upon but also covers the opposite eye. The virus of encephalitis confers immunity against herpes and vice versa. After an animal or a human has recovered from an attack of herpes, the conjunctival sac still carries the virus. Doerrs and Vochting found the virus entered the aquaeous but did not go into the vitreous. Gifford and Lucie introduced herpes virus into the ciliary body of one eye of a guinea pig and uveitis on both sides followed. From the second eye another animal was infected with similar results. Six per cent of the experimental animals developed herpes on the

opposite cornea. Autopsies upon these animals showed the route from the first to the second eye, by which the infection traveled, was along the optic nerve to the chiasm and then to the opposite eye. Various investigators have experimented with different aims in view. Some have tried to show a similarity of herpes with lethargic encephalitis, while others have had impetigo contagiosa, cowpox and chickenpox in mind. The experiments do not work out alike for all animals nor for the same animals with different experimenters.

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DISCUSSION.

Dr. S. B. Marlowe, Syracuse, N. Y.: The observation of four cases of herpes zoster ophthalmicus treated with intramuscular arsphenamin is my reason for entering this discussion. Dr. Lloyd has gone to some length in discussing the probable etiological factor for these conditions and the likely location of the lesion. The purely local and symptomatic treatment he has outlined is quite commendable. However, admitting the penetration of the virus and the deep situation of the lesion it would seem only reasonable that some form of medication might be effective if carried to the diseased part—the nerve root ganglion. Arsenic has long been recommended for these cases, usually in the form of Fowler's solution. Its effectiveness, however, has been slow and doubtful. Intramuscular arsphenamin, on the other hand, appears to be much more prompt and certain. Milian has reported five cases treated with arsphenamin with prompt relief. (Paris Medical 1-53-84.)

Four cases, all with corneal involvement, have been treated by this means in addition to the usual protective measures. The first case was seen in July, 1929. She came complaining of severe pain over her right eye which suggested sinus disease. She was sent to an otolaryngologist who opened the right antrum. The pain continued in spite of this, and shortly afterward the typical eruption appeared on the skin of her upper lid, the right side of her nose and forehead. Herpes zoster was diagnosed and the patient referred to an internist, Dr. W. A. Groat, who gave her an intramuscular injection of sulpharsphenamin (0.6 mgm.) In 24 hours the pain was much improved; in 48 hours completely relieved. The eruption on her skin cleared within a few days. There had been a large area of epithelial disturbance of the cornea, which healed eventually with an

insignificant scar and very little visual loss. Three other cases have since been treated in the same manner with similar results.

Although four cases is too small a number from which to draw final conclusions, the results obtained were so striking that it has seemed desirable to emphasize the value of this procedure. Experience so far has proved it to be time-saving; it may prove to be sight-saving.

Dr. J. P Corser, Scranton, Pennsylvania: I wish to discuss but one type of the disease under consideration, namely, recurring herpetic keratitis. The case histories of at least one form are singularly alike and are about as follows:

There is a sensation of foreign body in the eye increasing for 24 to 36 hours, with sensitiveness to light. The eye feels dry, but usually during the night at the end of the first 24 hours there is profuse lacrimation, intense photophobia, and the sensation of a large, sharp foreign body on the tarsus of the upper lid. Examination of the eye during the first stage shows a few dilated bulbar conjunctival blood vessels leading to one sector of the limbus, and in the corresponding sector of the cornea, usually near the margin, one or more small superficial infiltrated spots. When the lacrimation with increased pain and photophobia takes place, these spots stain with fluorescin. These attacks last from seven to twenty-one days and recur at intervals of one month to one year. During the interval the patients are hypersensitive to light, and use of the eyes for near is attended with more or less ocular irritation.

Of nine such cases occurring in our practice, two continued to have these attacks; one will not carry out instructions; six have now been over two years without recurrence. This has been brought about by the avoidance of certain foods, principally shell fish, port, buckwheat flour, and tea. It would seem, therefore, that allergy may be a factor in the etiology of this type of herpetic keratitis. The use of ephedrin by mouth or adrenalin hypodermatically has given marked relief from the symptoms and shortened the attacks.

Dr. E. E. Blaauw, Buffalo: Herpes corneae occurs more often than is suspected, and I have not seen any help from the slit lamp in its diagnosis. I speak only of the dermotropic form. The diagnosis is made with fluorescin eventually, with the addition of methylene blue.

As to recurrence, we have none. I like to think of the herpes virus as something like the pneumococcus, more or less ubiquitous, able to live in a healthy conjunctival sac, and giving trouble from time to time. Of course the eye should be closed, but only when we have destroyed the entire sick epithelium, and that is possible if you use tincture of iodine. I have used it for twenty-five years and have not seen a bad result. In most cases the epithelium is restored in two or three days. The patient will complain of much pain some time after the application of tincture of iodine, but this passes off and can be soothed with anodynes. If the cornea is deeply infiltrated it is remarkable how the infiltration disappears when the epithelium is restored, leaving hardly any scar tissue, following ordinary routine treatment.

In disciform keratitis the primary cause may be herpes corneae, but I suspect a secondary infection, which accounts for the necrosis of the substantia propria corneae.

REPORT ON INVESTIGATION OF LENS PROTEIN THERAPY IN CASES OF CATARACT.*

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An investigation has been conducted by the authors to learn of the clinical value of lens protein therapy in cases of cataract. Several different phases of the subject have been looked into.

1. The value of treatment of cases of senile cataract with lens protein injections.

2. The matter of production of lens protein anti-bodies in the blood serum of cases treated.

3. The influence of these injections on the patients when their cataracts are removed surgically.

4. Intradermal sensitivity of patients with senile cataract to lens protein.

5. The desensitization of such patients as are sensitive to lens protein.

A supply of optilen, an extract of beef lenses, was received from the Eli Lilly Company for the test.

A preliminary series of patients was treated with optilen Series No. P20852B, at Bellevue Hospital. They received subcutaneous injections three times weekly between January 15, 1929, and March 15, 1929. None complained of any severe reaction. The average dose was 2 cc., and the average total amount administered was 50 cc. The record book of this was unfortunately lost, and the exact amounts received by each patient are not known. The use of this series of optilen was discontinued, however, when three different vials were found to contain no coagulable protein when tested by adding a few drops of N acetic acid and a few crystals of salt and boiling. The trichloracetic acid and sodium tungstate and sulphuric acid tests showed only traces of protein. The Biuret reaction gave a brown precipitate, inconclusive for protein.

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EFFECT OF OPTILEN SERIES No. P20852B IN TEN CASES OF CATARACT AT BELLEVUE HOSPITAL.
UNCOMPLICATED CASES.

Case No.	Initials	Age	Sex	Vision Before Treatment	Vision After Treatment	Type of Cataract	Period from beginning of treatment to last observation	Changes in Refraction	Remarks
1.	E. M.	79	F.	20/70+ 20/40+	20/50+ 20/30—	Cortical sutures and vacuoles	3 mos.	O. D. + .75S O. S. none	Improved
2.	M. G.	79	F.	20/40— 20/70+	20/40 20/70	Cortico-nuclear with vacuoles	2 mos.	O. D. — 1.00 O. S. none	Stationary
3.	A. P.	59	M.	20/25— 20/20—	20/25— 20/25—	Cortical lamellae	5 mos.	None	R. E. stationary L. E. slight loss
4.	A. K.	79	F.	20/70— 20/40—	20/70 20/70+	Cortical sutures and lamellae	1 year	O. D. — .50S O. S. — .50S	Definite loss
5.	S. S.	54	M.	20/30+ 20/25+	20/25+ 20/25+	Cortical lamellae and sutures	1 year	O. D. + .50 Cyl. O. S. — .50 Cyl.	Optilen 1 cc. weekly for 3 months. Practically stationary

EFFECT OF OPTILEN SERIES No. P20852B IN TEN CASES OF CATARACT AT BELLEVUE HOSPITAL. COMPLICATED CASES.

Case No.	Initials	Age	Sex	Vision Before Treatment	Vision After Treatment	Type of Cataract	Period from beginning of treatment to last observation	Changes in Refraction	Remarks
6.	A. W.	65	M.	10/200 20/30—	10/200 20/30—	Cortical lamellae, sutures and vacuoles	1 year	None	R. E. central scotoma. Macula degeneration. Not improved.
7.	W. F.	60	M.	8/200 10/200	10/200 10/200	Senile punctate	2 mos.	None	Toxic amblyopia. Alcohol. Lens opacities not changed.
8.	J. F.	66	M.	20/40— 20/200	20/40+ 20/200	Cortico-nuclear sutures and nuclear haze.	1 year	O. D. — 1.00 S. O. S. none	Glaucoma Not improved.
9.	T. S.	70	M	20/30— 6/200	20/30— 6/200	Cortical vacuoles	2 mos.		O. S. old thrombosis. Central vein. Not improved.
10.	A. T.	66	M.	20/40+ 20/30	20/40 20/25	Cortical wedges and vacuoles	2 mos.	O. D. none O. S. + .50 S.	Diabetes. Optilen injections discontinued because of diabetic complications.

Summary of Cases Treated at Bellevue Hospital.

Ten cases of senile cataract, five of them complicated by definite ocular or general disease, were treated with optilen Series No. P20852B (Lilly). Of the twenty eyes of these patients, seven were slightly better in vision, nine were the same and four were worse. This may seem to be a definite favorable result from the injections, but from a knowledge that the ophthalmoscopic and slit-lamp microscopic appearances did not change and from a knowledge of the minor variations of vision of untreated cataract patients, the visual changes, inasmuch as they are not striking, are held to be not dependent on the injections. Four of the complicated cases were held good cases for objective observations, as they had definite lenticular changes that could be easily examined at subsequent visits.

The Effect of Optilen Series No. P22327 in Cases of Cataract.

The following cases were treated at Vanderbilt Clinic. An intracutaneous test for sensitivity to the protein was first made. They then received subcutaneous injections in the upper arm three times weekly. The first injection was of 1 cc., after which the doses were increased 1 cc. each time until 5 cc. was reached, and the succeeding injections were of this amount. Only two patients complained of unpleasant local reactions. No cases of anaphylaxis developed.

Case No.	Initials	Age	Sex	Vision before Treatment	Vision after Treatment	Type of Cataract
204743	M. W.	53	M.	20/40 20/40	20/40 20/40	Cortical Sutures
238471	C. H.	82	M.	20/100+ 20/40—	20/70— 20/40—	Cortical Sutures and Lamellae
240132	A. P.	69	M.	20/40+ 20/50+	20/40 20/50	Cortical Sutures and Lamellae
215597	S. P.	70	M.	20/40— 20/70	20/40— 20/100+	Nucleocort. Nucleus and Lamellae
238748	M. K.	62	M.	H. M. 20/40—	L. P. 20/40+2	Cortical Lamellae and Vacuoles
235267	M. A.	61	M.	20/70+ 20/200	20/50 20/200	Postcort. Vacuoles
232760	P. G.	69	M.	20/20— 20/40	20/25+ 20/40+	Cortical Sutures and Vacuoles
229530	A. Y.	58	M.	20/25— 20/25—	20/40+ 20/30	Cortical Lamellae
	A. S.	69	M.	20/40 20/100	20/40 20/100+	Cortical Sutures and Lamellae
242924	T. C.	66	M.	L. P. 20/30+	L. P. 20/30+	O. D. Cortico- nuclear O. S. Sutures and lamellae.
242230	M. O'T.	56	F.	20/30+ 20/30+	20/30+ 20/20—	Cortical Sutures
229732	E. M.	70	F.	20/70— 20/40	20/70— 20/30	Cortical Sutures and Lamellae
229103	V. M.	45	F.	20/50+ 20/70	20/50— 20/50—	Ant. and Post. subcapsular
217037	S. W.	64	F.	20/40 20/40—	20/40 20/40—	Cortical Sutures and Lamellae
241135	M. M.	62	F.	20/70 20/70	20/200 20/200	Ant. and Post. subcapsular
221467	F. B.	74	F.	20/30— 20/30—	20/30— 20/30—	Cortical Sutures and Lamellae
190598	D. B.	64	F.	20/100 20/70	20/70— 20/70	Nuclear
229596	E. W.	75	F.	20/40 20/30	20/40— 20/30—	Cortical Sutures and Lamellae
227775	E. H.	68	F.	20/40 20/40	20/40 20/40	Cortical Sutures
195549	J. S.	65	F.	20/40 20/40	20/40 20/40—	Cortical Sutures and Lamellae
247197	M. T.	54	F.	20/40+ 20/30	20/40— 20/40—	Cortical Sutures and Lamellae
247543	S. V.	74	F.	20/30— 20/30	20/40 20/30—	Cortical Lamellae
176496	S. B.	68	M.	20/70 20/50	20/200 20/50	Cortical Sutures and Lamellae
239057	S. M.	55	F.	20/40 20/200	Stopped Treatment Hemorrhagic Retinitis	Cortical Sutures and Lamellae

Case No.	Number of Injections	Total Quantity of Optilen	Duration of Treatment	Changes in Refraction
204743	17	65 cc.	3 mos.	O. D. no change O. S.—.50 Sph.
238471	11	49 cc.	3 mos.	None
240132	22	99 cc.	3½ mos.	O. D.+.50 S. O. S.+.50 S.
215597	24	102 cc.	4 mos.	O. D. no change O. S.—.50 Sph.
238748	17	75 cc.	2 mos.	None
235267	17	76 cc.	2 mos.	O. D. none O. S.—2.50 S.
232760	18	80 cc.	2 mos.	O. D.—.25 O. S.—.50
229530	20	87 cc.	3 mos.	No change.
	18	86 cc.	4 mos.	O. D.+.50 O. S.+.50
242924	18	75 cc.	2 mos.	None
242230	15	69 cc.	2 mos.	None
229732	14	64 cc.	2 mos.	O. D. no change O. S.+.50 Sph.
229103	19	80 cc.	3 mos.	No change.
217037	24	110 cc.	3½ mos.	O. D.—.75 c. O. S. no change
241135	14	60 cc.	2 mos.	No change.
221467	21	81 cc.	3 mos.	No change.
190598	17	75 cc. Optilen A	2 mos.	O. D. no change O. S.—.50 cyl.
229596	13	55 cc.	2 mos.	O. D.—.25 S. O. S.—.25 S.
227775	13	55 cc.	2 mos.	None
195549	18	84 cc.	2 mos.	None
247197	21	93 cc.	2½ mos.	None
247543	14	60 cc.	2 mos.	None
176496	11	35 cc.	3 mos.	O. D. no change O. S.—.50 Sph.
239057	11	45 cc.	1½ mos.	Not checked

Precipitin Test After Injections	Intradermal sensitivity to Parke-Davis Lens Protein Bovine	Remarks on Results
Neg.	Fleeting ± ½ cm. Reaction	Possibly slightly more haze in cortex and greater involvement of the sutures. No subjective improvement.
Neg.	Neg.	No improvement. Pt. complained of further failure of vision.
1-10 ± 1-100 Neg. 1-1000 Neg.	±	Pt. states "eyes feel stronger." Objective test shows no improvement.
1-10 Neg. 1-100 + 1-1000 ±	±	Not improved.
Neg.	.....	Not improved. Extraction of Right lens advised.
Neg.	Neg.	Not improved.
Neg.	.....	Subjectively "no better." Not improved.
1-10 + 1-100 Neg. 1-1000 Neg.	Fleeting ±	Subjectively "I feel about the same." Objectively slight progress of haze and lamellar separation.
Neg.	Neg.	O. D. no change. O. S. very slight improvement.
Neg.	+ + +	Not improved. Extraction O. D. performed.
Neg.	Neg.	"I do not see any difference." Ojectively some improvement in vision in O. S.
Neg.	Fleeting ± ½ cm.	Ophthalmoscopic and microscopic—no change. Improved from 20/40 to 20/30 in O. S.
1-10 ± 1-100 ± 1-1000 ±	Fleeting ±	"I see better and am able to work." Objectively not sufficient change to justify this statement.
1-10 + 1-100 + 1-1000 —	±	Not improved.
1-10 ± 1-100 + 1-1000 ±	.....	"Depressed concerning condition. Gotten worse recently." Opacities have progressed fairly rapidly.
Neg.	±	No change.
1-10 Neg. 1-100 Neg. 1-1000 ±	.....	"Sees no better." Objectively no improvement.
1-10 ± 1-100 Neg. 1-1000 ±	Neg.	"Health and sight better this year than ever before." Ophth. and microscopic appearance indicates progress in opacity.
Neg.	Neg.	No change.
1-10 ± 1-100 Neg. 1-1000 Neg.	.....	Practically no change.
Neg.	Neg.	"Feels eyes are better." Actually a visual loss.
Neg.	Fleeting 1 cm.	Diabetes under treatment. "Has not noticed any difference." The injections did not disturb her general condition.
.....	Neg.	Unable to continue injections. Arteriosclerosis. Hypertension and prostatic retention.
.....	.....	Nephritis. Hypertension. Developed hemorrhagic retinitis.

Results.

Twenty-four patients with senile cataract, eleven male and thirteen female, ages 45 to 82 years, were treated with optilen Series No. P22327 (Lilly). Eighteen were of the cortical type, one pure nuclear, two cortico-nuclear and three were post-cortical or subcapsular. They received intradermal tests with optilen Lilly Series No. P22327 (Lilly) and all who subsequently received subcutaneous injections were first determined to be not sensitive to the material used. The number of injections varied from eleven to twenty-four and the total amounts administered ranged from 49 to 110 cc. The period of treatment varied from two to four months. The patients were regular in attendance, their attitude was good and they were very much interested in getting results. Twelve patients expressed definite opinions in regard to the results. Four were positive their eyes were better, but objectively, two of these were the same and the other two showed a loss. Six patients said they were about the same, while two were depressed concerning their condition, saying that they were worse. The objective results were judged by comparison of the vision with best correcting lens accepted before treatment and the vision under the same conditions after operation. If the vision was estimated under cycloplegia before treatment, the same was done afterwards. If by manifest refraction, it was done in like manner. The ophthalmoscopic appearance, as judged with the aperture of the instrument and the slit-lamp microscopic appearance, were also used to aid in the judgment of any objective changes in the lens opacities.

I. Vision.

Forty-eight eyes of twenty-four patients were tested before treatment and re-tested after treatment. Eight eyes had better vision, twenty had the same, and twenty had poorer vision. The changes for the better or the worse were not great in any case. It is well known that the vision of patients with cataract varies from day to day, even with apparently the same conditions. The results are not due to changes in refraction or to better or worse refraction.

Changes in Refraction.

It is a well known fact that the development and progress of lens opacities entail changes in refraction, often very startling.

These latter have been proved to be due particularly to changes in the nuclear portion of the lens. Cortical cataracts may progress considerably without causing great changes of refraction. From a consideration of these facts, one might expect that if a case of nuclear cataract is improved or arrested, there might be a loss of some of the lenticular myopia or that it at least should stop in its progress. Of the seventeen cases of cortical cataract in which the refraction was checked after the treatment, twenty-three eyes had the same focus, five were slightly more hyperopic and six slightly more myopic. Of the three cases of post-cortical cataract, five eyes were of the same focus and one had become slightly myopic. Of the three cases of nuclear and nuclear-cortical cataract, four eyes were the same, while two had become slightly more myopic.

Ophthalmoscopic and Microscopic Appearance.

In not a single case was there observed any definite improvement in the appearance of the lens, as judged with the aid of the ophthalmoscope or slit-lamp microscope.

Precipitin Test.

Unfortunately these were not performed before the treatment was started. After the injections, thirteen patients were entirely negative for anti-lens precipitins in their blood serum, six were just plus-minus and must be counted negative, while two were positive in one dilution of antigen and one was positive in two dilutions and two were not tested. Of the three cases showing these immune bodies in the blood serum, two were females who had received totals of 110 and 60 cc., respectively, for the 2 + and the 1 + reactions and one male who had received 102 cc. of the optilen Series No. P22327 (Lilly). Apparently this did not affect the intradermal sensitivity, as in none of the + precipitin patients was there found intradermal sensitivity. In only one patient of those who received the optilen injections was there found pronounced sensitivity to the intradermal use of the lens protein. His precipitin test was negative. His case is reported in a following section.

Comments on Results.

Of the twenty-four cases under treatment, no striking results were obtained. A few cases showed slight improvement, but the great proportion were just the same or worse. Subjectively, some

said they were better, but in these cases their statements were not justified, as in some of them their vision was worse. As judged with the aid of the ophthalmoscope and slit-lamp microscope, none of the patients was improved. For any non-surgical treatment of cataract to be judged efficacious, there must be some cases definitely improved, as with ordinary hygienic measures many cases can be held stationary for a period of years. It may be said that the amount of lens protein solution given in this series was too small. But 5 cc. is about as much as can be conveniently given in individual doses three times a week for a period of several months. The patients stood these injections well. The patients who have received the injections are known and will be recalled to the clinic at intervals in the future so that their future reactions may be known.

Discussion.

One may be permitted to discuss the possible value of lens protein therapy in cases of cataract.

1. Which portion of the lens is represented by solutions of lens protein usually used for injection? Saline or aqueous solutions commonly used contain portions of alpha and beta and gamma crystallin, usually not the insoluble albuminoid. These crystallin proteins represent the clear transparent lens material.

2. Are anti-lens immune bodies produced? Yes, for they can be demonstrated in some cases by the precipitin test and by anaphylactic reactions.

3. Do these reach the aqueous and lens? It has been demonstrated that the ciliary epithelium acts as a semi-permeable membrane, and because of its selective action holds back practically all immune bodies. It is only when the ciliary epithelium is injured or diseased or degenerated that such bodies pass.

4. Granting that the epithelial membrane is degenerated to some extent in cases of cataract and the anti-lens bodies enter the aqueous and lens, what effect would they have on the lens? Such immune bodies are definitely specific, and if any reaction occurred it would be with the remaining clear, transparent portions of the lens which represent the remaining alpha, beta and gamma crystallins. If the reaction is as is produced in the test-tube, that of specific precipitin reaction with antigen used, then precipitation of these transparent proteins which are in solution would result and further opacity would be produced.

5. Can reaction between the specific anti-bodies and the denatured protein of existing lens opacities occur? No, because of the specific nature of the anti-bodies.

6. Can lens protein therapy act as a specific tonic for the lens? No, it may act as a foreign protein and stimulate the body defenses and reactions in general and then secondarily affect the lens, but not primarily act as a tonic for the lens, for the reasons given above.

Reactions of Patients Who Received Optilen to Surgical Removal of Cataracts.

The sensitizing action of lens protein therapy in patients with cataract and the possible effect at time of the operative removal of the cataracts has been a matter of discussion. Five patients who received subcutaneous optilen Series No. P22327 (Lilly) injections came to operations afterwards.

T. C., male, aet. 66, received 18 injections for a total of 75 cc. of optilen Series No. P22327. Uveitis developed after operation. See special report following.

M. A., male, aet. 61, received 17 injections for a total of 76 cc. of optilen Series No. P22327. Moderate amount of cortex remaining. No undue reaction and no uveitis after operation.

S. S., male, aet. 47, received 9 injections for a total of 35 cc. of optilen Series No. P22327. Moderate amount of cortex remaining. No undue reaction and no uveitis after operation.

S. B., male, aet. 68, received 11 injections of 35 cc. of optilen Series No. P22327. No undue reaction and no uveitis after operation.

M. K., male, aet. 62, received 17 injections of 75 cc. of optilen Series No. P22327. No undue reaction and no uveitis after operation.

Other patients of the series who received lens protein therapy are coming in for operations. Their results will be reported at a later date.

Intradermal Sensitivity to Lens Protein.

Thirty-eight patients with senile cataract were tested intradermally with lens protein (bovine) Parke Davis and two reacted positively. These two cases were particularly interesting because the mild uveitis which was present following their capsulotomy extraction subsided rapidly after subcutaneous injections of lens protein.

REPORT OF TWO CASES OF MILD PHAKO-ANAPHYLACTIC UVEITIS.

Case 1.—T. C., male, aet. 66 years, with mature senile corticonuclear cataract in the right eye and immature opacity in the left eye. Intradermal test with optilen Series No. P22327 were negative on February 27, 1930. From February 28, 1930, to May 9, 1930, he received 18 subcutaneous injections of optilen Series No. P22327, totaling 75 cc. The precipitin test taken on June 6, 1930, was negative for anti-lens bodies, showing 1/10 negative; 1/100 ±; 1/1000 negative. On June 2, 1930, he had a preliminary iridectomy performed on the right eye, and on June 25, 1930, the cataract was removed by the capsulotomy method. A moderate amount of cortex remained and a moderate reaction in the form of an uveitis persisted for five weeks after operation. On July 30, 1930, the intradermal test with Parke Davis lens protein (bovine) was performed and was positive, the area of redness and elevation of the skin being 1½ cm. in diameter and persisting for ten days while the control saline injection was negative. On August 4, 1930, he was given ½ cc. of Parke Davis lens protein (1-10) subcutaneously. There was no systemic reaction. This dose was repeated on August 11, 1930, and on August 18th, 1 cc. of the (1-10) protein was administered. The uveitis began to subside soon after the first injection and all congestion and aqueous disturbance had disappeared within two weeks.

COMMENTS ON CASE NO. 1.

The intradermal test with optilen Series No. P22327, containing lens protein, was negative before the subcutaneous injections of optilen. Still, after these and after the cataract extraction it was found positive. Either he was sensitized by the injections or by his own lens protein. The improvement in the uveitis which followed the densitizing doses was most probably dependent upon these, although this cannot be proved. There are undoubtedly mild cases of phakoanaphylactic or phakogenetic uveitis such as this case that will respond to desensitizing doses of lens protein.

Case No. 2.—J. M., aet. 61 years, was first seen January 7, 1929, when he had O. D. 20/100, O. S. 20/200 vision with correcting lenses. In June, 1929, he had capsulotomy extraction of cataract from left eye after preliminary iridectomy. For two months following he had a mild uveitis which finally, after the moderate amount of cortex which remained had absorbed, subsided. Fol-

lowing discission of a thin secondary membrane, he obtained excellent vision, 20/25 +. On March 4, 1930, he had capsulotomy extraction of the cataract from the right eye with a greater amount of cortex remaining. It was not until May 2, 1930, that the true nature of the condition was determined as a mild phakoanaphylactic uveitis. The intradermal tests with Parke-Davis lens protein (bovine) and with optilen Series No. P22327 (Lilly) were markedly positive. The precipitin tests for anti-lens bodies in the serum were all negative. On May 7th, he was given subcutaneously 1 cc. of optilen Series No. P22327 (Lilly) as the first desensitizing dose. On May 9th, 2 cc. optilen; May 12th, 2 cc.; May 14th, 3 cc.; May 19th, 2½ cc. In less than two weeks the mild uveitis, which had been treated locally for two months, had subsided. On August 27, 1930, discission was performed with good results.

Comments on Case No. 2.

This case is likewise a case of mild uveitis following cataract extraction. The intradermal sensitivity which was demonstrated and the rapid response to the desensitizing injections, which contained very small amounts of lens protein, are part proof of the nature of the condition. However, the spontaneous disappearance of the uveitis from the fellow eye previously, would detract some from the effect of the optilen as being the specific agent in clearing up the inflammation.

Summary and Conclusions.

An investigation has been conducted to learn of the clinical value of lens protein therapy in cases of cataract.

Optilen Series No. P20852B (Lilly) was used in ten cases of cataract at Bellevue Hospital. The results were such that conclusions could not be drawn. However, by tests it was discovered that this series of optilen contained such extremely slight traces of protein that its use was discontinued. The amount present was negligible in the matter of possible production of antibodies.

Optilen Series No. P22327 (Lilly), which was tested and found to contain considerable protein, was then used in twenty-four cases of cataract at the Vanderbilt Clinic. Intradermal sensitivity tests were first performed. The results were judged according to:

1. Changes in vision.
2. Changes in refraction.
3. Changes in ophthalmoscopic appearances.
4. Changes in slit-lamp microscopic appearances.

From a consideration of all the factors, the conclusion was drawn that the minor changes that occurred in these factors in these patients during the period of optilen therapy were not produced by the injections.

The precipitin test was performed on twenty-two patients after the optilen injections. Thirteen were entirely negative for anti-lens precipitins in their blood serum, six were plus-minus and must be counted negative, while two were positive in one dilution of antigen (both 1/100), and one was positive in two dilutions (1/10 and 1/100).

Five patients came to the surgical removal of their cataracts after having received optilen injections. Four of these had no undue reaction after capsulotomy extraction and were not sensitive to lens protein, while the fifth, who was not sensitive before operation, became sensitized afterwards and developed a mild phakoanaphylactic uveitis. This responded well to desensitizing doses of Parke-Davis lens protein (bovine).

One other patient developed spontaneously a mild phako-anaphylactic uveitis after capsulotomy extraction. Desensitizing injections of optilen Series No. P22327 apparently helped clear up the uveitis in the second eye operated on, even though it is recognized that the uveitis in the first eye cleared up without desensitizing injections.

It will be well to limit these conclusions to the cases under observation only and not attempt any generalizations.

OTOLARYNGOLOGIC SESSIONS

NON-SPECIFIC PROTEIN THERAPY.

JOSEPH C. BECK, M. D.,

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When I accepted the instructions to present this paper before the Academy, I determined upon three definite courses:

1. That I would be very sympathetic to the subject of protein therapy, knowing full well that the future of serology and immunology in all its possibilities is destined to play a great rôle in medicine.

2. That since the application of protein therapy was general or systemic rather than local, and since this systemic condition, whether toxic or septic, was due to some local pathological process, I would be insistent upon surgical removal of such local pathology, and not treat it by any other method. This, of course, must be qualified by the possibility of contraindication to any kind of operation or specific medical treatment.

3. That I would avoid, as much as possible, repetition of well known facts, thus relieving the already overburdened literature on this subject.

Foreign protein therapy divides itself into two great headings:

1. The Theoretical and Research.

2. The Practical or Clinical Application.

It is the latter, the clinical, that I shall practically limit myself to and only sketch for you the progress that has been made along the theoretical and research part of the work, depending upon the gentlemen who have favored me with their willingness to discuss my paper to further elaborate upon that side of the subject. (Slide 1.)

AUTHORS OF NON-SPECIFIC PROTEIN THERAPY IN GENERAL.

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G.—Weichardt, W.: Non-Specific Protein Therapy as a Means of Increasing Healing Processes.

Ergebnis d. Hygiene, Bakter., Immunitetsforsch u. exp therapie, Vol. 5, 525, 1922.

H.—Wolff, L. H.: Non-Specific Therapy. Zeit. Exper. Medicine, Vol. 67, 5683, 1929.

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Peterson, who has published the monograph on Non-Specific Therapy in 1922, is constantly quoted in every article on this subject, and I have made use of that book and several later publications of his in order to be informed.

It was Wright in 1912 (?) who brought out his opsonic index and the specific vaccine therapy, which was followed by a very enthusiastic response in its application, and the literature swarmed with articles on this subject. However, in 1923, he changed his viewpoint and accepted that much of the specificity of bacterial products was really active on a non-specific basis and gave it the term "epiphylaxis."

Recently an article appeared from the pen of Dr. Joseph Miller in the Journal of the American Medical Association, entitled "The Present Status of Non-Specific Therapy," and in his introductory remarks he refers to a personal conversation with a surgeon, about twenty years ago, concerning specific vaccine therapy and its shortcomings. I would also like to refer to that time, when I presented to this Society a paper on Wright's opsonic index and specific vaccine therapy in chronic suppuration of the nasal accessory sinuses and middle ears. It was a negative report, as far as cures were concerned, and most of these cases were later operated on. Since then much better indication for vaccine or protein therapy has been established, as well as better technique in its application. (Slide 2.)

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Foreign protein therapy does not mean that only a foreign protein substance such as (refer to Slide 3):

PREPARATIONS USED IN NON-SPECIFIC PROTEIN THERAPY.

A. Milk and Milk Preparations.
   1. Aolan
   2. Caseosan
   3. Novoprotin
   4. Phlogetan
   5. Terpichin
   6. Omnadin
   7. Caseal
   8. Lactosan
B. Yatren
C. Yatren-Casein
D. Strepto-Yatren
E. Stomasin of Centanni
F. Alfasol (vegetable protein)

Must be introduced into the system *per se*, for it is the belief today that as a result of even specific sera or chemicals, such as neosalvarsan, mercury, quinine, etc., foreign protein develops in the system and through them specific antibodies develop which in turn destroy bacteria, protoza, or neutralize the toxemia, thus curing the disease. Certain definite reactions are expected to result when a foreign protein is introduced into the system or when it develops as a result of the introduction of bacteria, chemicals, or even physical agents, such as radiation, X-ray, radium, quartz-light, etc.

Ferdinand Hoff, in his monograph, which deals with this subject most scientifically and interestingly as regards these definite reactions, shows a table that, while it refers to reactions of infections of short duration, is found applicable in most instances where foreign protein is acting on the body. It is divided into two phases. (Slide 4.)

HOFF'S TABLE OF REACTIONS.

PHASE 1.	PHASE 2.
1. Rise in temperature	1. Drop in temperature
2. Leucocytosis (myelocytic)	2. Drop in leucocytosis with a tendency to a lymphocytosis
3. Decrease in alkali reserve (acidosis)	3. Increase in alkali reserve
4. Increase in basal metabolism	4. Minus basal metabolism
5. Increase in albumosis	5. Less albumosis in the blood
6. Increase blood sugar	6. Decrease blood sugar
7. Decrease of cholesterin in the blood	7. Increase in the quantity of cholesterin
8. Hyperactive sympathetic nervous system	8. Hyperactivity of the parasympathetic nervous system
9. Increase in thrombocytes (blood platelets) and their destruction	9. Thrombocytes return to their normal proportions

Locally, at the site of injection, we may have considerable swelling, especially in cases of certain bacterial vaccines, such as pyocyaneus. However, in the majority of substances that are employed very little reaction is found at the point of injection.

I have not seen a reaction locally at the site of the chronic lesion which is reported by some writers.

Alexander speaks of a case of mastoid disease, wherein he employed a foreign protein therapy that caused such a reaction and destruction of the mastoid as could not have occurred from the disease itself, and it was the occasion of his being compelled to operate much earlier than he wanted to. It is true that he employed foreign protein therapy in acute cases, whereas, we have only employed it in early chronic and chronic cases

The various pathologic processes about the ear, nose and throat in which we have employed foreign protein therapy, and, of course, that includes all the cases of earlier times when we used autogenous vaccines, or the following (Slide 5):

1. Otitis externa
    - A. Eczematoid
    - B. Follicular, or furunculosis
2. Otitis media chronica
    - A. Catarrhalis
    - B. Suppurativa
3. Postoperative mastoid disease both after simple and radical mastoid

4. Chronic sinusitis
   A. Non-suppurative
   B. Suppurative
5. Postoperative sinusitis both after simple or conservative or more radical operations.
6. Atrophic rhinitis
7. Chronic tonsillo-pharyngitis, with or without acute intercurrent attacks
8. Chronic laryngo-tracheitis, especially suppurative
9. Bronchiectasis
10. Borderline cases, such as:
    A. Chronic suppurative salivary duct disease or any of the salivary glands
    B. Fistulæ about the head and neck

As to the actual number of cases treated, it is surely irrelevant. Suffice to say that a large number (several hundred) have received the treatment in the past ten years and the following conclusions are drawn therefrom (Slide 6):

CONCLUSIONS.

1. Although acute pathologic conditions have been subjected to foreign protein therapy, the results, in our hands at least, have not been encouraging.

2. While anaphylactic shock has not occurred in our series, its possibility should be borne in mind and a careful history should be taken, especially as to previous use of biological sera, and this particularly in children. Protein injection should not be given unless adrenalin is at hand for emergency use.

3. Clearly indicated surgery was not permitted to be superseded by non-specific protein therapy, and when it was employed in systemic contraindication to surgical intervention its use was not attended by any noteworthy benefit.

4. The chill that has been supposed of prime importance in non-specific protein therapy was rarely observed in our cases.

5. Other therapeutic measures, such as specific sera, vaccines, allergins, chemicals, physical agents, diet and endocrines also possess definite non-specific protein effects as adjuvants in our therapeutic attack upon disease.

6. This conclusion I do not care to publish, and that is that too many men employ non-specific protein therapy on the advice and

information of commercial salesmen of the various milk and bacterial, etc., products without the necessary laboratory and scientific medical control. This can only lead to the disrepute of the treatment.

DISCUSSION.

DR. BURT R. SHURLY, Detroit: The subject of nonspecific protein therapy is of particular interest to the clinician and research worker because it is in this field of biochemistry and research that the hope of valuable achievement becomes more fascinating with encouragement. The future longevity and health of the race can be tremendously influenced for the better by increased success in the fight to maintain and fortify the cell with an increased resistance to infection. The danger to the profession and public is the tendency of some temperamental minds to become so easily affected by witchcraft, voodooism and bunk, as handed out by the unscrupulous vendor of shots, that the genuine scientific truth is given jolt after jolt until our faith is shaken.

This essayist has dealt fairly with this problem of useless and unscientific therapy that sways the profession in waves of belief in and doubt of newer methods of medication. He hits the nail on the head when he says he would insist upon the surgical removal of local pathological conditions wherever indicated. I would go further and demand that the new, untried and experimental be left in the laboratory or research hospital where it belongs, or added only after surgery and specific treatment with methods of reputation have failed in their application. When surgery and successful older methods of therapeutics are exhausted, additional aids have their place. Nonspecific protein therapy belongs to this type.

Wright's opsonic index and vaccines have failed to deserve the overenthusiasm with which they were heralded to an ever ready profession and public.

I wish to call to your attention the splendid bibliography that accompanies the essayist's paper.

Milk and its preparations, introduced hypodermically, are well known to the ophthalmologist. In suppurating processes in the ear, nose and throat many claims are made. The effect I believe is transient, although I do not find in the literature a long series of carefully observed cases. The value of foreign protein introduced into the system, with the attending development of specific antibodies that raise resistance to infection, is well established. The importance of raw vegetable juices such as those of carrots, tomatoes, beets, celery and potatoes, and especially vitamin A, in the growth of the cell and resistance to infection, is well recognized. It is only necessary to call attention to the toxic and lethal effect of the frequent injection of eggwhite into the animal body, and to study Hoff's table of reactions to realize the profound effect on the biochemistry of the cell.

It is well known in the experimental feeding of white rats for vitamin A deficiency, that beyond 28 days xerophthalmia develops in 80 percent

or more of the animals. The symptoms of corneal ulceration and blindness, drying of the lachrymal apparatus, infection and suppuration of the antrum and middle ear, ulceration of the tongue and respiratory infection, are demonstrated in various research laboratories. Control rats with 5 drops of codliver oil do not develop the disease.

If nonspecific protein therapy is to control disease in the same remarkable manner that codliver oil controls xerophthalmia, it is necessary to separate and concentrate the essential food protecting factors. This has been done for codliver oil and we have irradiated ergosterol under various names in dosage of 3-5 minims. In the Detroit College of Medicine Research Laboratory we are now in the midst of an elaborate demonstration of the curative effects of preparations of concentrated vitamin A made from carrots in oily solution; also vitamin A from liver dissolved in fish oil. Experiments will be conducted on diluted portions of the oils to determine a correct dosage, and to estimate the true value of a concentrated vegetable protein that experimentally raises resistance and rapidly modifies the symptoms of xerophthalmia.

Dr. Samuel Levinson, Chicago: Dr. Beck's paper on nonspecific therapy is extremely interesting. I should like to emphasize the last conclusion which Dr. Beck stated, namely, the promiscuous use of various preparations in the field of nonspecific therapy and various clinical conditions, particularly in ear, nose and throat. We must not lose sight of the fact that therapy in general, and particularly nonspecific therapy, should not be a routine form of treatment, but an individualized therapy. We must take into consideration the resistance of the patient, and the degree of stimulation produced by the drug. From the clinical standpoint, therefore, we must consider that the nonspecific reaction is a diphasic reaction, the first effect being the intensification of the disease process both locally and generally. The second phase of this reaction is a constructive phase, in which there occurs a general euphoria and a diminution of disease symptoms, both general and local, and at times a restitution to normal. This diphasic reaction depends on the stimulation of the cells, and beneficial results cannot be obtained if the organism is not capable of responding to stimulation. Therefore, the usual response to nonspecific therapy should be stimulation and a greater cellular activity, and if this stimulation is continued the cell becomes fatigued, leading to complete exhaustion and death. It is obvious, therefore, that in order to produce the most beneficial results proper dosage must be used, so that one gets a transient stimulation with changes in metabolic processes which become clinically manifest by increase in weight and a general wellbeing of the patient. Dr. Peterson emphasized the important point which has also been mentioned by Dr. Beck, that nonspecific therapy should be an individualized therapy and never a routine, and that the dosage, preparation and time of application should be varied according to the disease and the resistance of the patient.

Dr. Beck discusses Hoff's tables of reactions, and we should like to show the relationship this classification has to Dresel's vagotonic and sympatheticotonic status of individuals. In Hoff's tables the reactions are divided between those whose blood pressures are high versus the low

blood pressures, the differences in the basal metabolism, the carbondioxide combining power of the blood, and the calcium content. These differences in the reactions of the individuals to nonspecific therapy can be divided into two classes: one, the vagotonic status, which shows that the tissues are alkaline, contain little calcium colloid and much potassium colloid; and the blood is alkaline and contains little ionized calcium, much bound calcium and little potassium. This is contrasted with the sympathetico-tonic status of the individual, in which the tissues are acid and show much calcium colloid with little potassium colloid; and the blood is acid and contains much ionized calcium, little bound calcium and much potassium.

Dr. Edwin McGinnis, Chicago: I have not had much experience with nonspecific protein therapy except in association with the Eye Department at the college and hospital, where I have been much impressed by the beautiful results they have gotten in the treatment of acute iritis, and in the Medical Department where they have been using it in polyarthritis. These are two conditions in which nonspecific protein therapy acts almost like magic.

A number of years ago I had the pleasure of visiting in Boston where I saw a good deal of the work of Doctors Goodale and Walker in the diagnosis and treatment of hayfever. On returning to Chicago, after playing with ragweed extract and various extracts of pollen, I gleaned a good deal of information from the injection of specific therapy—pollen extracts—for the cure and relief of hayfever. I had some very striking allergic reactions. One case I remember was a woman about 35 or 40 years old who was a severe hayfever victim. I had been treating her with ragweed extract for six or eight weeks, and she could finally take the strongest solution we had without getting a reaction. During the course of the treatments she had an acute cold, and the next dose of ragweed extract gave her a terrific allergic reaction. It was a sight I never will forget. Her eyelids were swollen, her arms, hands and limbs became swollen and edematous, her skin was shiny red, and she was having respiratory difficulties. Adrenalin fortunately relieved the symptoms and her difficulties were over for that time.

In the treatment of acute iritis, relief of the pain comes within 24 to 36 hours after the injection of typhoid vaccine. The redness of the conjunctiva continues, however, for two or three weeks. In working with the Eye Department we have examined these patients intranasally, and in most of the cases we have been able to make a diagnosis of acute anterior ethmoiditis. I have been interested for a good many years in the surgical treatment of acute ethmoiditis and I have found that a ditch drainage of the anterior ethmoidal cells gives a better result in the iritis cases, in that pain is relieved in about 24 hours, while the redness and congestion of the eyeball clears up in less than five days. These patients are not so apt to have recurrences, but if they are so unfortunate as to have a recurrence after a cold we are able to shorten the second attack by shrinking down the lining membrane of the anterior ethmoidal cell walls, and the iritis is promptly relieved.

Dr. Frank J. Novak, Jr., Chicago: Dr. Beck's excellent paper serves a good purpose because it will stimulate interest in the subject of im-

munology. I believe it was Pasteur who said that the future physician will be an immunologist.

We know a great deal of the results of the clinical application of nonspecific protein therapy, but we know very little of the mechanism of the reaction. If you noticed the table Dr. Beck showed—Hoff's table—you will find the first phase shows a leukocytosis. Most men do not agree with that. Kolmer has shown that in the first phase there is leukopenia, and later a leukocytosis. As to the mechanism involved in nonspecific protein therapy, it is generally believed that it is simply cell activation.

There is a great list of things that can be used as nonspecific proteins. Some time ago I tried to make a list of the various substances that have been used in nonspecific protein therapy, and after covering five or six pages with handwriting I gave it up. Among other things it is interesting to find that the use of pilocarpin increases the formation of antibodies in the production of diphtheria antitoxin.

Here is an interesting fact: the individual who has received the antigen, say typhoid, and then later is injected with nonspecific protein—it may be a serum or it may be an exudate—that individual produces a large amount of antibodies specific for whatever disease he may be having. But if he has not been sensitized first with the antigen, there is practically no increase in the antibodies. The question arises, Where do these antibodies come from? Of course, that is the big problem of immunology. I do not think there is much question at present that it is from the reticulo-endothelial system. The cells of the reticulo-endothelial system are activated by the specific injection. These cells are found in the spleen as well as in the liver, the omentum, and the bone marrow, and it has been shown that after injection with specific therapy there is increased activity of the cells of these particular tissues.

As to dosage, a great deal of damage may be done by the use of nonspecific protein therapy at the wrong time and in improper dosage, and also, the injection of foreign protein at a time when the cellular mechanism is incapable of further stimulation. We must proceed carefully in the use of this treatment.

Dr. Joseph C. Beck, closing: My hope has been realized, because the gentlemen who have discussed the paper have really made it. The scientific part, which was practically all that was touched upon in the discussion and is of great value, brought out one fact, that these substances are not to be used as a placebo, and that they may do great harm if they are used by inexperienced individuals and without proper control. We will never stop a great deal of the indiscriminate use of injurious substances, but I have an idea since we do not see the great trouble from the use of these commercial products, that perhaps it is just a placebo that we are giving rather than nonspecific therapy. Take raw milk, for instance, and commercial milk, and boil them the same time, and you will find a great difference in the protein reaction from these two milks, because the older milk, the infected milk, contains bacteria that are destroyed by boiling, while the commercial protein is infected and therefore the greater reaction will result.

Dr. Novak touched upon the reticulo-endothelium, which is the second defense mechanism, and it is not only in the parts of the body he mentioned, but all through the entire system. Examination of the blood will show it. Perhaps the blood is not examined early enough and the leukopenia is not found. It is usually leukocytosis in the early type and the myelocytes are increased, and in the second stage there will be a decrease, or if there is an increase it is the lymphocytes and not myelocytes that are found.

There are many points in Hoff's table, or in any table, showing that it will be necessary to have cooperation between the laboratory man and clinician in order to make use of this splendid means of therapeusis.

THE EMBRYOLOGY OF THE LARYNX.

ROBERT F. RIDPATH, M. D., F. A. C. S.,

PHILADELPHIA.

The larynx mechanically, anatomically, physiologically, neurologically and pathologically has been the subject of innumerable essays, monographs and voluminous treatises, and yet this little organ, one and one-quarter inches of the human body, defies the analytical minds of our greatest medical thinkers in some of its mechanical and neuroanatomic-physiologic action.

It is not my intention to dwell on the anatomic description as usually found in the various textbooks, but, on account of the exceedingly limited amount of literature on the subject of embryology, to bring before this society the embryologic phase of the subject.

In preparing to write, I found I must in the first place ignore the ordinary accepted theory that the larynx is the "voice box." This thought is forcibly brought to our attention when we realize the many species of animals having larynges similar in all respects to the human and who do not use this mechanism for voice or speech.

We are therefore brought to a pointed question, viz.: if the larynx was not originally intended for vocal production, what was its original usages and how has it developed into the organ we know?

From the division of a fertilized ovum forming daughter cells a multicellular embryo results. In general, the development of an embryo depends first upon the multiplication of its cells by division; second, upon the growth in the size of the individual cells, and third, upon changes in the form and structure of the cell division.

In general, all cells multiply from other cells in one or two methods of cell division—amitosis and mitosis.

In amitosis, the cells divide directly by the simple fission of their neuclei and cytoplasm; in mitosis, the reproduction necessitates complicated changes in the nuclei.

The first arrangement of the cells seems to be in the three primary germ layers, named from their position the ectoderm (outer layer), the mesoderm (middle layer) and the entoderm (inner layer).

"These cells of the germ layers are at first alike in structure. Gradually the ectodermal cells multiply, change their form and structure and give rise to the layers of the epidermis."[1] By more structural changes the mesodermal cells are changed and transformed into the elementary cartilage, bone, muscle, connective tissue, etc.

Cells which have once taken on the structure and functions of a given tissue cannot give rise to cells of any other type. In other words, a line of specialization once begun cannot be transformed into another type; this gives us the law of genetic restriction.

As development advances, there is a constantly increasing restriction in the kind of differentiation open to the various parts. Each distinctive tissue or organ is more rigidly bound to its specific type of cells than was the material from which it sprang. The original or parent tissue is likewise limited by being unable to duplicate analogs already formed. Thus, the primary cricoid can never become anything but a cricoid; whereas, the gut from which it originally sprang, at other levels, buds off the lungs, liver, pancreas, etc. But if the embryonic cricoid were destroyed the pharynx or gut could never replace it. In other words, to repeat, whenever specialization once begins, whether it be connective tissue, muscle or what not, there can be no transformation or reversion to another type.

The youngest human embryo of which we have knowledge is the Miller specimen. It is similar to the diagram as represented in Fig. 40a. Other specimens are the Byce teacher embryo, as in Fig. 40b and Fig. 40c. These ova all belong to the latter part of the second week. In the third week the ovum possesses a distinct primitive groove and allantois. (Fig. 32a.) A head process with its notochordal canal features the advanced state, illustrated in Fig. 32b, in the Ingall embryo. There is also noted at this time the beginning of the neural groove.

In Fig. 57, we notice that the specimen has considerably progressed. This is Spee's specimen. The primitive streak is confined to the end of the embryonic disc. This is about the third week.

Embryos of the fourth week are characterized by distinct, definite neural folds. (Fig. 58.) The edges soon unite along part of their extent to form a tube which is the *anlage* of the brain and spinal cord. (Fig. 59.)

Embryos of the fifth week reveal external swellings below the primary brain vesicles which develop into the future neck; these swellings, separated by grooves and depressions, form the brachial arches. The first pair bifurcates and forms the upper and lower mandibles. Between them a depression appears, the future mouth. (Fig. 91.)

Embryos of the sixth to eighth week show marked changes. Externally their form resembles more the adult type, and after the second month the embryo is designated as a fetus.

The Branchial Arches.—The formation of the larynx "involves the history of the branchial arches. These are barlike prominences separated by grooves which occur on the lateral surface of the neck."[1] They correspond to the gill-bearing arches of the fishes.

The human embryo develops five arches, separated by four ectodermal grooves; subjacent to the grooves the ectoderm of the pharynx bulges correspondingly. (Fig. 87.)

Toward the end of the sixth week, the first and second arches overlap one another and the other three obscure them. Fig. 63 shows the beginning of this process; Fig. 227, an advanced stage, and in Fig. 64 it is complete. See Fig. 84, A, B, C.

In embryos of five to six mm., the oval end of the laryngeal groove is bounded on their lateral sides by two prominences, the arytenoid swellings, which are connected with a transverse ridge, forming the furcula of His; this ridge becomes the epiglottis and is derived from the third and fourth brachial arches.

These arytenoidal swellings assume a curved shape later and form the posterior walls of the glottic opening, which at this time is T-shaped. Although the future glottic opening is closed at this time, the epithelium soon dissolves with the withdrawal of the arytenoid swellings backward and the forward movement of the epiglottis, leaving an opening which soon after the tenth week becomes oval in form. (Fig. 106.)

Before the formation of the oval larynx we detect the formation of the ventricles and shortly after we are able to visualize the true vocal cords.

The Cricoid.—The cricoid *anlage* can be distinguished toward the fifth week of embryonic life as a concentration in the fifth

arch. "They are really deviations from the trachea and represent the more primitive form of larynx."[2] "During the following week or the 14 to 16 mm. embryo the cartilages, although existing purely as condensed mesenchyme, have developed into distinctly outlined masses and assume their more or less adult relationship. The cricoid is at first an incomplete dorsad crescent, but eventually forms a complete ring."[3]

In the seventh week each half of the cricoid cartilage is partly cartilaginous in front and below and has closed completely posteriorly. The rate of growth during the latter end of the second month is more pronounced than at any other time, following which it, the growth, is decidedly slower. During the eighth and ninth week, the chondrification is practically complete; the anterior-inferior ends are turned slightly down before they join, to form a notch. During the tenth to eleventh week this notch is practically eliminated.

Arytenoids.—These cartilages are derived from the fifth arch and are closely applied to the laryngeal cleft. Early concentration occurs during the sixth week, more prominent than the cricoid concentration from which it is slightly separated; these masses grow more slowly.

"Standing up behind the end of the central mass with the accompanying upgrowth of the adjacent fourth arches, they make the back wall of a transversely deposed cavity, the laryngeal cavity, above the true cords."[4]

By the beginning of the third month the shape of the ultimate arytenoid is fairly well marked and chondrified. The vocal processes still lack chondrification and we are forced to the conclusion that the process is separately chondrified and fusion later takes place with the larger mass of the arytenoid.

The Thyroid.—At the end of the fourth week a small rudiment of this cartilage is seen as a mass of closely packed cells immediately in front of the fourth pouch and cleft. "It is a condensation of the fourth arches on each side of the fifth arches, the two halves chondrify and join ventrally early in the third month."[4] It is fairly possible that the cartilage contains some elements of the fifth arch, but if this is so it is added afterwards. Each plate has two centers of chondrification.

About the sixth week the concentration of the thyroid is continuous with the hyoid. About the eighth week chondrification

has progressed considerably. The anteroinferior angle is quite pronounced and overhangs the cricoid, and a suggestion of the inferior cornu is observed. The alae are now seen to be in direct contact, with the exception of the superior-anterior portion where the incisura is indicated.

The Epiglottis.—The cartilages of the epiglottis appear relatively late and it is not until the early part of the third month that we find the mass of small concentrated cells in the central or pre-cartilage mass which constitutes this cartilage.

It increases in size slowly and grows from three points, a central and two lateral or wing concentrations. This cartilage is different from the other cartilages in that it is elastic while they are composed of hyaline constituents. "The corniculate cartilages represent separate portions of the arytenoids."[4] The cuneiform cartilages are derived from the cartilage of the epiglottis.

The Muscles.—Toward the end of the fifth week, four pre-muscle masses can be recognized, two on each side of the embryonic larynx. The masses are entirely separate or independent of each other. The upper group consists of the cricothyroideus, cricoarytenoideus lateralis and the thyroarytenoideus.

In the lower mass the cricoarytenoideus posterior is well differentiated.

No connection or apparent relation with the pharyngeal constrictors exists.

During the sixth week, the muscular elements have grown with considerable rapidity, and at the seventh week have received fibrillation and can be readily differentiated from each other.

"About the beginning of the eighth week all the muscles are clearly represented and capable of absolute differentiation."[3]

I have tried to make clear that all the laryngeal musculature develops independently and not as a sphincter, a view formerly held by Strazza and others.

The laryngeal muscles originate in the fourth and fifth branchial arches.

The Nerves.—Simliar to the pre-cartilages and pre-muscular masses, so the nerves can be differentiated as early as the fifth week of embryonic life. At this time the recurrent laryngeal nerve has pushed upward as far as the cricoarytenoideus muscle.

"During the sixth week the nerves have distinctly advanced and can readily be traced to their terminations, and the anastomosis

between the inferior and superior laryngeal nerves has been accomplished."[3]

By the end of the seventh or early eighth week the nerve supply of the musculature is complete.

The vagus nerve, of which the recurrent is a branch, develops from the fourth and fifth branchial arches.

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DISCUSSION.

Dr. Ralph A. Fenton, Portland, Oregon: It is necessary to realize what prodigious labor has gone into Dr. Ridpath's splendid presentation, which has the very special merit that it represents the opinion of a busy clinician, directed toward a technical problem which has long been relegated to microscopists, often without medical background.

Detailed embryologic study, especially of the complex innervation of the larynx, may prove of great value in the study of so-called hysterical aphonia, as well as in evaluating predisposing factors toward the various palsies. Irregularities in the development of neural connections, always more likely where primitive organs take on complicated new functions, await explanation from Dr. Ridpath's continued study.

Vulnerability of the cords to new growths, benign or malignant, and the arytenoid and epilgottic region to the implantation of microorganisms, may be found to depend upon anatomic factors arising far back in the embryonal development of these structures. Their degree of local resistance will be found, as elsewhere in the body, to depend not only upon contemporary trauma or exposure, but very decidedly upon the extent to which Nature has built good materials into the larynx and its adnexa. Wherever a structure has swerved from its early physiologic, involuntary object to take over functions under the dominion of the will, numerous neurologic and degenerative disorders become likely. Careful embryologic analysis thus may throw much light upon such tendencies toward laryngeal disease in the child or adult.

Dr. John F. Barnhill, Indianapolis: What has been said by the essayist is so clear and represents such simple facts that one cannot discuss these facts alone; they are not to be discussed. It occurs to me, however, that one might make a few useful observations, because unless one can do this, can deduce from such a paper as this some practical points, it would fall by the wayside.

I therefore wish to make the observation, based solely upon my own experience, that the larynx is remarkably free in adult life from any thought of derangement connected with its embryology. In the neighborhood, in the environment of the larynx we find several affections in which

Nature seems not to have done a complete job in embryology. For instance, in the upper branchial arches we find a cause for cleft palate and harelip, and in the lower arches we find a similar cause for defects, so called branchial defects, which are sometimes very difficult to remedy. We may have a branchial cyst or a fistula which is thoroughly explained by the embryology. But embryological defects do not seem to exist when we come to the larynx itself. There are few if any defects in the larynx which we see in adult life which we believe can be wholly attributed to any embryonal defect. It might be assumed that certain abnormal appearances, as for instance a large thyroid cartilage, is of embryonic origin. It is not certain, however, that it is a defect; it may be apparent only, and due to the life of the individual, to a very cylindrical, long neck, or to a very prominent Adam's apple.

The embryological defects of the larynx are in my judgment of less frequent occurrence than in other tissues of the human body.

Dr. Thomas E. Carmody, Denver: Very little can be said in discussion of this paper except to compliment Dr. Ridpath on the work he is doing. I feel that this paper, along with the brilliant work that has been done by our confrere, Negus of London, on comparative embryology and anatomy of the larynx, are two steps in the right direction from the standpoint of clinical observation. I feel with Dr. Fenton and Dr. Barnhill that the work by clinicians will tell us very much more than the work by embryologists and anatomists has ever done—giving them due credit, however, for having laid the foundation for us clinical men.

From the standpoint of deformities, Dr. Barnhill has spoken of upper deformities, and these almost all take place in the first few weeks of embryonic life. As Dr. Ridpath said, the larynx practically begins its development at three weeks, and the main part of it is complete by the end of the eighth week. This also applies to the mouth, pharynx, tongue and other associated parts. Consequently we have here something that if kept in mind will explain possibilities of infection and of injury, whether early in life or later.

Dr. Robert F. Ridpath, closing: I have nothing to add except to thank the members who have discussed this paper for their complimentary words. I hope to present the original work that I am doing on the larynx in this line at a later meeting.

THE DIAGNOSIS AND TREATMENT OF ALLERGIC DISEASE OF THE NOSE AND PARANASAL SINUSES.*

FRENCH K. HANSEL, M. D.,

ST. LOUIS, MO.

The diagnosis and treatment of allergic disease of the nose and paranasal sinuses entail the consideration of the various disorders designated as hay fever, vasomotor or hyperesthetic rhinitis, bronchial asthma, catarrhal rhinitis and sinusitis, non-suppurative hyperplastic nasal and paranasal sinus disease, hyperplastic ethmoiditis, nasal and sinus polypi and edemas. It is now generally conceded that with few exceptions these diseases are the manifestations of allergy or human hypersensitiveness.

The subject under consideration is so extensive that it is possible to give only a brief outline of the important phases. The proper understanding of this subject entails not only a thorough knowledge of the clinical symptoms and pathologic changes but also requires a knowledge of the general subject of allergy.

In previous studies[1 2 3 4] I have already described in detail the clinical and histopathologic phases of nasal and paranasal sinus allergy and have emphasized the importance of differentiating the allergic diseases from the primary inflammatory or suppurative processes in the nose and paranasal sinuses.

For the present, therefore, consideration will be given to the general outline of a method suggested for the routine investigation employed in establishing the diagnosis and the general principles of the treatment of the nose and sinuses and the treatment of the allergic state of the patient. In the Department of Otolaryngology of Washington University Medical School and Barnes Hospital we have followed this routine in the investigation of all cases of nasal allergy. The investiga-

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tion and treatment of the allergic state of the patient is conducted in the Asthma Clinic, under the direction and cooperation of Dr. H. L. Alexander and Dr. C. H. Eyermann.

The clinical and histologic pictures are not always classical, for consideration must be given to the fact that the patient may be temporarily free of allergic manifestations for a variable period of time. The temporary cessation of nasal symptoms may frequently be accounted for by the fact that the patient is affected with some other manifestation of allergy, such as hives, eczema, or angioneurotic edema. It must also be remembered that the occurrence of acute infection or the establishment of chronic secondary infection frequently complicates the diagnostic problem.

In order to establish the diagnosis of allergic disease of the nose and sinuses a routine method of investigation should be adopted. It is suggested, therefore, that the following factors be considered: (1) nasal symptoms, (2) rhinoscopic examination, (3) examination of nasal secretions, (4) Roentgenographic study of sinuses, and (5) histopathologic examination of nasal and sinus tissues. A study of the allergic state of the patient should be made with particular reference to: (1) The occurrence of other manifestations of allergy, (2) a positive family history of allergy, (3) the presence of positive skin tests, and (4) blood eosinophilia.

Briefly the nasal manifestations of allergy are characterized symptomatically by sneezing, nasal obstruction and nasal discharge. In hay fever these symptoms are frequently well marked and easily recognized. In the other, or nonseasonal types, the symptoms are generally intermittent, vary considerably in degree and are apt to be confused with frequent colds.

The nasal discharge in allergy varies from thin serous liquid to thick tenacious mucus and the microscopic examination usually shows the presence of eosinophiles.

The examination of the nose in allergy reveals pathologic changes which are usually strikingly characteristic. The membrane appears discolored grayish pink or bluish gray, with swelling or edema. Edema is particularly apparent in the middle meatus or anterior ethmoidal regions. In cases with more advanced pathology, hyperplasia and polypoid formations may be observed. The nose may be filled with polypi which are attached usually in the anterior and posterior ethmoidal regions.

These formations are the result of sagging or prolapse of the edematous mucosa. The examination of the sinuses, particularly the ethmoidal cells and the maxillary antrums, may show edema, hyperplasia and polypi as found in the nose.

In the histologic examination of the nasal tissues the average mucosa contains a certain relative amount of connective tissue, lymphocytes, other lymphoid cells, eosinophiles, blood vessels, cavernous vessels, glands, lymphatic and distended interstitial spaces. Finck[5] has shown that in definitely abnormal states the nasal mucosa presents relative increase or decrease in these tissue elements under the epithelium, that is, in the tunica propria or stroma. The epithelium may show thickening, hyperplasia or loss of substance.

The histopathologic examination of the nasal and sinus tissues in allergy shows pathologic changes similar to those found in the bronchial tissues in bronchial asthma. The surface epithelium is thickened and consists of many layers of stratified columnar cells. Certain areas may be observed where thickening is not sufficiently marked to cause loss of cilia. Areas of desquamation of epithelium appear scattered here and there. The subepithelial tissues are loose and edematous and infiltrated with eosinophiles which are usually found in the tissues in allergy. Marked accumulations frequently occur just under the epithelium. Other cellular elements consist mostly of plasma cells and lymphocytes. Large tissue spaces contain serum which gives the edematous appearance. The glandular structures usually appear dilated and filled with secretion. Large cystic areas lined with epithelium represent dilated glands or gland ducts. In advanced stages atrophy and disappearance of glands may occur. Cellular accumulations appear around blood vessels and glands. On the whole, the cellular reaction is much less but the edema is more marked than that seen in true inflammatory processes. The typical pathologic picture of allergy is confined chiefly to the subepithelial layer of the mucosa and usually extends partly into the glandular layer. Edema and eosinophilic infiltration rarely extend deeper than the glandular layer. The number of eosinophiles in the blood, the tissues and consequently also in the nasal secretions may show variations in different cases. Even in the same tissue specimen there may be considerable variation in the distribution of cellular elements. Eosinophiles may be scarce in some areas and very densely collected in

other areas. Similar variations occur in the distribution of lymphocytes and plasma cells. Edema may be more pronounced in some areas than others. Variations in the cellular elements, exclusive of the eosinophiles, may be dependent upon the age of the patient, the occurrence of frequent attacks of acute infection or upon the chronicity of the allergic process.

Bone changes are confined, practically, to the ethmoidal cells where there is a tendency to rarefaction. Bone changes here are frequently absent and when present are relatively insignificant unless well pronounced.

Roentgenographic examination of the paranasal sinuses in allergy frequently shows varying degrees of opacity, depending upon the degree of pathologic change and the presence or absence of retained secretions. Edema and polypi may be shown by the use of lipiodol and affords a much better method of interpretation of the actual pathology present. Edemas in the sinuses may be acute or chronic. Acute edema occurring in early or even chronic cases is frequently transitory, giving positive findings at one time and negative at another, or vice versa. Proetz[6] recently reported these findings in two cases and has most comprehensively illustrated his findings. Similar cases have been observed by Dean[7] and Johnson.[8]

In addition to a careful investigation of the nose and paranasal sinuses as just outlined, a most careful and painstaking history must be undertaken to further establish the diagnosis of allergy. A complete history will not only determine the existence or nonexistence of the allergic state, but quite often will disclose the etiologic factors. The family history is of utmost importance in strengthening the diagnosis. The existence of asthma, hay fever, urticaria or eczema in the antecedents or in the brothers and sisters or in the patient's children strongly suggests that the patient is allergic. Spain and Cooke[9] and Cooke and Vander Veer[10] found that 53 per cent of 966 cases of allergy gave a positive history of allergy in the antecedents. Balyeat[11] reported a history of 60 per cent in one thousand cases of allergy. Adkinson,[12] Rackemann[13] and others have reported on the hereditary influence of allergy and found a positive history in more than 50 per cent. In the investigation of 115 normal individuals, Spain and Cooke[9] noted a history of allergy in only 7 per cent, while Balyeat[11] in like studies found 9 per cent in 403 normal individuals.

In taking the history of the patient himself, certain facts may be outstanding in establishing the diagnosis. The age of onset is important because it is frequently noted that the various manifestations of allergy make their appearance early in life. Eczema, urticaria, frequent colds, bronchitis often occur in infancy and childhood and suggest the existence of allergy.

Occupation frequently determines the causative factor and this should always be thoroughly investigated. There are numerous occupations in which the individual comes in contact with various allergens, and it is important to consider these substances as etiologic possibilities.

An exhaustive study of the patient's environment should be made regarding the contacts in the home. This should include the character of bedding, carpets, furniture, the presence of house pets, house flowers, and the use of face powders, sachet powders, talcum, etc.

The dietetic history is also of great importance, for the part played by foods is being proved of increasing significance. The patient should be questioned regarding the foods eaten, the frequency with which they occur in the diet and their possible relation to symptoms. The patient should keep a diary of the foods eaten so that they may be considered in relation to symptoms.

It is quite impossible to go into further detail regarding the importance of a good history. Such a history requires thoroughness, a comprehensive knowledge of the subject and the intelligent cooperation of the patient.

Further evidence of the existence of the allergic state may be obtained by the performance of skin sensitization tests. The positive skin test merely indicates the presence of the allergic state and does not of necessity develop the etiologic diagnosis. It is possible that the allergic state may exist in the presence of negative skin tests. The skin may be positive or negative to certain substances which cause nasal symptoms. It is unnecessary to test the patient with many substances with which he has no contact. The clinical, environmental, occupational and dietetic history often suggest or indicate the probable offending allergic agent.

The eosinophilic reaction is generally recognized as a phenomenon of allergy, but not as a definite indication of its existence, for it is known to occur in association with other diseases.

Eosinophiles occur not only in the blood but in the nasal and sinus secretions. They do not always run parallel in the blood and nasal secretions, for I have found them in the secretions in great numbers, while the number in the blood was normal. Failure to demonstrate eosinophiles in the blood and nasal and sinus secretions should not be construed that allergy does not exist. There may be periods when the patient is temporarily free of symptoms, or may have been entirely free of nasal symptoms for a long period of time. In many cases of nasal polyps of long standing, in which the patient is not actively allergic, there may be an absence of eosinophiles.

In addition to a thorough investigation of the nose and paranasal sinuses, therefore, the diagnosis of allergy is further based upon a detailed clinical history, dealing with the presenting symptoms, the antecedents, the home environment, the occupation and the diet of the patient, the results of skin sensitization tests and the demonstration of eosinophiles.

The treatment of allergy of the nasal and paranasal sinuses may be divided into: (1) The treatment of the allergic state of the patient, and (2) the treatment of the nose and paranasal sinuses.

In the treatment of the allergic state of the patient many drugs are employed to give symptomatic relief. The effect of adrenalin, particularly in asthma, is well known. Ephedrin, atropin, calcium, potassium iodide, cocain and other drugs are also employed.

Prophylaxis is undoubtedly the most important phase of the treatment of the allergic patient. The prevention of vasomotor attacks in the nose and paranasal sinuses gives more satisfying relief than any other form of treatment. By prevention or marked alleviation of reactions, advanced structural changes in the nose and paranasal sinuses are prevented and the occurrence of secondary infection becomes less probable. If early cases without definite polypoid changes can be controlled, the edema present frequently disappears and the subsequent pathologic changes may be avoided. Every resource should be employed to discover the offending allergen.

As already mentioned, a careful and painstaking history often discloses the offending factors and gives important clues leading to the cause of attacks. While there are always many possible allergens which may cause symptoms, in the majority of cases,

there are usually only a few allergens responsible for symptoms. There are certain allergens which stand out as the most frequent and important offending substances. It is usually customary, therefore, to consider first, such agents as feathers, household pets, house dust and the use of face powders and other toilet articles. Skin tests are performed with the suspected allergen and if positive, strong suspicion that this is an offending factor is justified. Although a positive skin reaction does not necessarily indicate that the particular substance is causing symptoms, each reacting allergen should be considered and investigated to prove whether it does or does not cause symptoms.

Offending foods may be discovered by placing the patient on special diets, such, for example, as those suggested by Rowe.[14] By these diets the most common offending substances, such as wheat, milk, eggs, raw and uncooked vegetables, condiments, etc., are excluded and the diets made up of foods which are not commonly eaten or which usually do not cause reactions. If the patient becomes free of symptoms, various foods are then added until symptoms are produced and the offending agents are discovered. The patient is instructed to keep a diary of every food eaten and to record all symptoms. Skin tests may be done with the suspicious allergens.

Food allergy as a cause of nasal symptoms has been thoroughly and comprehensively studied by Eyermann.[15,16] He emphasized the fact that the allergens appearing most frequently in the diet are the ones that most frequently cause the various clinical types of nasal allergy, and that some cases of pollinosis may be rendered comfortable merely by food restrictions. He also states that many cases of perennial vasomotor rhinitis may secure satisfactory clinical results without densensitization injections. Food and inhalants should be considered as additional factors in hay fever. Balyeat,[17] Cohen and Rudolph[18] and others have also emphasized the importance of considering multiple sensitization in hay fever. Eyermann[16] has shown that in many of these cases it appears that the intensity or the duration and possibly also the initiation of symptoms depend upon the total allergic dose.

Frequently it is necessary to employ desensitization treatment in cases when it is difficult or impossible to remove or to avoid the offending allergens. This applies particularly to such agents, for example, as pollens, orris root and horse dander.

Nonspecific agents are frequently used in treatment but give only temporary results. Such agents are tuberculin, peptone, vaccines and other substances such as milk, sulphur, etc.

In the treatment of the nose and paranasal sinuses in allergy several drugs such as adrenalin, ephedrin, cocain, etc., are used to give temporary relief.

In the surgical treatment of the nose and sinuses in allergy each and every case presents an individual problem. Surgical interference is usually based upon the degree of pathologic change present and upon the presence or absence of secondary infection. At best any surgical operation will not cure the patient of his allergy. Operations should be performed, therefore, either to eliminate infection or to restore function as best possible. Submucous resection, removal of polypi or edematous swellings are justified to restore nasal respiration. Radium may be employed to lessen or to prevent the recurrence of polypi. Operations on the sinuses are often necessary to eliminate infection, particularly in the maxillary sinuses. Too much reliance should not be placed upon X-ray findings, for, as has been proved, edematous swellings in the sinuses may be transitory, or if permanent may not be doing any particular harm to the patient.

The presence of edema in the sinuses, particularly in the antrums, is not in itself an indication for operation. Antrum puncture is frequently necessary to determine the presence or absence of infection, the retention of secretions or for the purpose of introducing lipiodol. Removal of the middle turbinate and exenteration of the ethmoid cells is frequently performed in allergy cases, but it has been my experience that these operations are rarely ever necessary, and may do much harm to the nose in destroying its function and subjecting the lower respiratory tract to greater irritation.

In any event, operations upon the nose and paranasal sinuses should be conservative, for, after all, the allergy is the most important factor and must be controlled if satisfactory results are to be obtained.

Allergy as an etiologic factor in nasal and paranasal sinus disease has become one of the most important problems in otolaryngology and must of necessity become a part of it. The rhinologist must, therefore, adopt not only a systematic method of studying the nose and paranasal sinuses but must also acquire an adequate knowledge of the general subject of allergy. The

diagnosis of allergy from the standpoint of the otolaryngologist will frequently disclose the existence of other important manifestations of the disease. Certain migraine headaches, for example, have been proved to be definitely of allergic origin and have been relieved by appropriate treatment of the allergy. Gastrointestinal symptoms such as nausea, vomiting, gas eructations, diarrhea and abdominal pain are not infrequently associated with nasal allergy. Certain cases of Ménière's syndrome have been proved to be of allergic origin. Allergy is increasing in importance in all fields of medicine. The rhinologic diagnosis of allergy may be, therefore, an important factor in disclosing the allergic origin of some other disorder.

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The following outlines were presented as lantern slides:

DISEASES CONSIDERED AS BEING OF POSSIBLE ALLERGIC ORIGIN

1. Pollinosis or Hay Fever.
2. Vasomotor or Hyperesthetic Rhinitis.
3. Catarrhal Rhinitis and Sinusitis.
4. Nonsuppurative Hyperplastic Nasal and Paranasal Sinus Disease.
5. Hyperplastic Ethmoiditis.
6. Nasal and Sinus Polypi and Edemas.
7. Combined Suppurative-Hyperplastic Sinus Disease.
8. Bronchial Asthma.

ROUTING METHOD OF INVESTIGATION (RHINOLOGIC).

1. History of Nasal Symptoms.
2. Rhinoscopic Examination.
3. Nasal Secretions.
4. Roentgenographic Study of Sinuses.
5. Histopathology of Nasal and Sinus Tissues.
6. Antrum Puncture and Antroscopy.

STUDY OF ALLERGIC STATE OF PATIENT.

1. Detailed Clinical History:
   a. Family History.
   b. Occurrence of Other Manifestations of Allergy.
   c. Presenting Symptoms, Environment, Diet, etc.
2. Skin Sensitization Tests.
3. Eosinophilia.

CLINICAL HISTORY.

1. Family History:
   a. Antecedents.
   b. Brothers and Sisters.
   c. Children.
2. Other Manifestations of Allergy:
   a. Pollinosis or Hay Fever.
   b. Urticaria or Hives.
   c. Eczema.
   d. Asthma.
   e. Angioneurotic Edema.
   f. Certain Other Disorders, such as:
      1. Headaches.
      2. Mucous Colitis.
      3. Nausea and Vomiting.
      4. Abdominal Pain and Diarrhea.
3. Personal History:
   a. Presenting Symptoms, Seasonal or Nonseasonal.
   b. Age of Onset.
   c. Symptoms Early in Life.
      1. Eczema, Urticaria, Asthma.
      2. Colds and Bronchitis.
         a. Febrile or Afebrile.
      3. Food Intolerance.
      4. Skin Rashes.
4. Contacts:
   a. Occupational.

1. Bakers, Farmers, Millers, Cooks; Grain Dusts, Flour, etc.
2. Druggists, Dentists; Powdered Drugs.
3. Farmers, Equestrians, Dairymen, Laboratory Workers, Veterinarians; Animal Emanations.
4. Wood Workers, Leather Workers and Dyers.

b. Environmental:
1. Bedding and Down Comforts; Feathers.
2. Mattresses and Furniture; Horse, Cattle, Rabbit, Camel and Goat Hair.
3. Furs, Pillows and Toys of Children; Cat and Rabbit Hair.
4. Clothing and Scarfs; Wool, Camel Hair.
5. Cosmetics; Orris Root, Rice Powders, Perfumes, Sachets, Hair Tonics and Dyes.
6. Occupation of Other Members of Household.
7. Animals, Cats, Dogs, etc.
8. Household Plants, Insecticides, Dust.
9. Proximity to Factories, Stables, Granaries, etc.

c. Dietetic:
1. Food Intolerances.
2. Quantity and Types of Food Eaten.
3. Relation of Foods to Symptoms.
4. Other Manifestations from Foods, Urticaria, Eczema, etc.

TREATMENT.

1. Treatment of Allergy:
a. Prophylaxis:
1. Inhalants; Animal Emanations, Drugs, Pollens, Dusts, etc.
2. Foods; Wheat, Egg, Milk, Chocolate, Stringbeans, Potato, Pea, Fish, Tomato, Onion, etc.

b. Desensitization:
1. Pollen, Horse Dander, Orris Root, House Dust.
2. Foods; Wheat, Egg, Milk.

c. Nonspecific Therapy; Tuberculin, Peptone, Vaccines, Milk, Sulphur, etc.

2. Treatment of Nose and Sinuses:
a. Symptomatic, Drugs.
b. Surgical Operations:
1. Submucous Resection.
2. Removal of Polypi and Edema.
3. Radium.
4. Removal of Middle Turbinate.
5. Ethmoid Exenteration.
6. Antrum Operations.
7. Frontal Sinus Operations.
8. Ethmosphenoid Operations.

3. Skin Sensitization Tests:
a. Negative Reactions.
b. Positive Reactions.
c. Most Common Allergens.

3. Eosinophilia:
Blood.
b. Nasal Secretions.
c. Tissues.

DISCUSSION.

DR. L. W. DEAN, St. Louis: It is very essential for the exact diagnosis of nasal lesions, especially of sinus disease, that a nasal smear be prepared and stained for the purpose of cytological examination. If this examination is not made I feel that all the information necessary for proper diagnosis of the case has not been secured. Certainly when pus cells are found in abundance in the nasal smear it means something; if nothing but mucus is found, it has a different significance; if an abundance of eosinophils are present, it suggests an allergic condition.

The method of making the examination is very simple. A cotton tipped nasal probe is passed into the nose. Touching the anterior nares is avoided if possible. It is desirable that the discharge in the neighborhood of the ostium of a suspected sinus be touched with the cotton on the probe. An ordinary microscopical slide is then smeared. A smear is prepared from each side of the nose. These smears are stained with Wright's or Giemsa stain, and the cells present studied.

The following case will illustrate the practical significance of this procedure. Last summer I saw a little girl whose nasal discharge contained 40 per cent. eosinophils. The remaining cells were epithelial cells and lymphocytes. There were only a very few polymorphoneutrophils. The general examination and history of the case all suggested that we were dealing with an allergic sinusitis. A few days ago, at the request of the allergist, I saw this patient. The inclement weather of the fall had produced more nasal disturbance than she had had during the month of August. The first thing I did was to make a nasal smear. Myriads of pus cells were present. Immediately we knew we had something different from that which we were dealing with in the summer. In short, we now had, in addition to the allergic sinusitis, a suppurative sinusitis. In this and other cases allergy must be considered as an etiological factor in sinus suppuration.

Several years ago Doctor Daniels showed that a diet deficient in fat soluble vitamin A caused sinus suppuration in the white rat. Wolbach and Howe have shown that there is a hyperplasia of the epithelial cells and a keratinization of these cells in these animals on a deficient diet before infection appears. At Washington University we have gone a step further and have shown that in these animals there are changes in the cells which precede the hyperplasia and the keratinization.

Deficient diet, allergy and other factors decrease the resistance of the cell to infection. An important consideration in every infection is this lack of immunity. Allergy, deficient diet and what not, must be given the greatest consideration when our therapy is applied.

DR. FREDERICK T. HILL, Waterville, Maine: I wish to express my appreciation of this very thorough paper on a difficult problem which is so often unsatisfactory, no matter how extensively we try to carry out the studies. The chief point that appeals to me is the conservatism manifested in the paper in regard to surgical treatment. There seems to be a tendency in many published articles to lean too far toward surgery in the treat-

ment of these cases. It seems it might be safe to say that surgery is advisable only where there are definite indications for it. So often we find that only temporary improvement follows any surgical operation in these cases. The work of Rackemann and Tobey showed that many patients that are hospitalized and not operated underwent the same improvement as did the operated patients.

I would like to ask Dr. Hansel how much significance he places, in regard to etiology, on the question of prenatal sensitization, that is, a food allergy which may be due to dietary indiscretions on the part of the pregnant mother.

Dr. Frank J. Novak, Jr., Chicago: I think Dr. Dean in his discussion touched upon a very significant and extremely important fact. He emphasized the point that we have to consider not only inflammatory conditions produced in the sinuses by the allergic conditions, but also the deficiency diseases and those classified under metabolic disturbances. Two years ago I presented before the Academy a paper in which I brought out a series of cases of vasomotor rhinitis, which is, I believe, fundamentally a metabolic disease. Since that time nothing has occurred to disprove, at least to my mind, that such is the case. A large number of patients have been studied since that time and certain observations have been consistently made. One is that these patients have a slow pulse, and a tendency to subnormal temperature; they show a tendency to vascular hypotension; they have a lowered metabolic rate. Upon the administration of thyroid these conditions change, the pulse becomes faster, the blood pressure goes up, the basal metabolism reaches normal, and most of the symptoms disappear.

I think, if I did not misunderstand Dr. Hansel, that he said there are types of hyperesthetic rhinitis which are not allergic in character. I am of the opinion that the great majority of hyperesthetic cases are incidental to a hypothyroid state. I wish I had the privilege of examining more closely the histologic specimens which the Doctor showed, but from where I sat I thought the appearance of the basal membrane and the edema of the connective tissue is the edema which one finds in the skin and mucous membrane in cases of hypothyroidism.

Dr. French K. Hansel, closing: I am glad to hear Dr. Dean emphasize the importance of the examination of nasal secretions. It is not unusual to see cases which appear to be acute rhinitis but which by careful observation and examination of the nasal secretions prove to be nasal allergy. This is particularly true in noses which show very little pathological change. The mucous membrane appears sometimes slightly pale and at other times slightly red.

As Dr. Dean emphasized, it is also important to take into consideration that the changes in the epithelium and the oedema which occur in allergy may be the factors which predispose the sinuses to infection.

Dr. Hill brought up the question of prenatal sensitiveness. We know that hypersensitiveness is inherited, and some observations have been made which quite conclusively prove that hypersensitiveness can exist prenatally.

Dr. Novak referred to the occurrence of slow pulse in the absence of hypertension in hyperesthetic rhinitis. It is generally known that hypotension is frequently found in the allergic patient. In our hyperesthetic rhinitis cases we have found practically all of them true to type, that is, we generally find eosinophils in the nasal secretions, and we believe that this is one of the chief indications that this condition is a manifestation of allergy. We have found very few cases with typical symptoms of hyperesthetic rhinitis which could not be proved to be of allergic origin.

THE SIGNIFICANCE OF THE SQUAMOUS EPITHELIUM IN THE CAUSE AND REPAIR OF CHRONIC MIDDLE EAR DISEASE.*

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This paper is presented for a twofold purpose: To give a more rational explanation of the findings in, and the course of, chronic suppurations of the ear and to depict the rôle of the squamous epithelium in the causation and attempted cure of these lesions.

The subject of this paper is based primarily upon the accepted principles of development laid down by Wittmaack[1]. These deal, first, with the influence of the various types of infantile otitis on pneumatization of the mastoid process and, second, with the bearing of infantile otitis upon the type and course of later otitic suppurations. In order to elucidate more clearly what follows, it is advisable to give a brief summary of Wittmaack's work.

According to him a pneumatized mastoid process is the only normal anatomical condition. Any other condition of the mastoid process is abnormal. In other words, the diploetic and the sclerotic mastoid processes are disturbed developmental changes and not variations of the normal. At birth the middle ear, the attic and the antrum are filled with a high type of embryonal connective tissue which covers the bony walls. This in turn is covered by a delicate layer of epithelium. The mastoid process, like all other bones of the skull, is a spongy bone and has between its bony trabeculae spaces which are filled with normal bone marrow. As soon as aeration takes place, the high embryonal tissue is subjected to atmospheric pressure and undergoes contraction with a certain amount of fibrosis. From the antrum this high myxomatous type of connective tissue, which is termed the subepithelial tissue, grows into the adjoining marrow spaces of the mastoid process; and this growth, in the normal, continues until all the marrow spaces have been replaced by this connective tissue. As air is permitted to enter the mid-

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dle ear and the connective tissue is subjected to the atmospheric pressure, it recedes and causes the middle ear and antrum space to enlarge and become air containing. In the spaces which border on the antrum and which have previously been filled with the embryonal connective tissue, there also occurs a contraction of this connective tissue; and with this contraction there follows a sinking of the delicate epithelial covering. After contraction has fully taken place there then remains, lining the bony trabeculae, only a thin layer of connective tissue covered by epithelium. Thus the spaces in the mastoid process, which formerly were marrow containing, are now in direct communication with the antrum and middle ear and have become air containing. This process is the normal process of pneumatization. To summarize, it consists of three stages: first, the ingrowth of subepithelial connective tissue into the marrow spaces, second, the contraction of the subepithelial tissue, and third, the sinking in of the epithelial covering.

Any disease of the middle ear, which occurs in infancy before the process of pneumatization has taken place, will cause a disturbance of this normal phenomenon. Aschoff[2] was the first to demonstrate the type of otitis media neonatorum which is due to the presence of meconium or another foreign substance which may have found its way into the middle ear during the early stage of respiration. This type has been termed by Wittmaack the catarrhal type of infantile otitis. It is a foreign body irritation and it causes, not a suppuration, but a localized destruction of areas of the epithelium in the middle ear, with a subsequent hyperplasia of connective tissue. This hyperplastic condition of the connective tissue is kept up by the foreign body irritation and ceases only when the foreign particles have been absorbed or encapsulated. Due to this hyperplasia, there never occurs the normal contraction of the subepithelial embryonal type of connective tissue. Consequently the connective tissue which has already invaded the marrow spaces in the mastoid spongiosa never undergoes contraction and there results a cessation of pneumatization at the end of the first stage. In other words, contraction of the connective tissue with sinking in of the epithelium does not occur. What then follows is a metaplastic bone formation within the spaces in the mastoid process which are filled with hyperplastic connective tissue. This results in what we commonly term a sclerotic mastoid process. The other type

of otitis media neonatorum is the suppurative type. Here a purulent process is present within the middle ear due to bacterial infection. After the cessation of the suppurative process, the inflamed mucosa of the middle ear and antrum is healed by fibrosis. Consequently there occurs a rapid and marked contraction of the subepithelial connective tissue. In these cases, the type of mastoidal structure found in later life, will depend upon the stage to which the process of pneumatization had advanced within the mastoid at the time of onset of the infantile suppuration. For instance, if the suppuration sets in before the first stage of pneumatization, no ingrowth of the subepithelial connective tissue into the marrow spaces in the mastoid process occur. We will then have throughout life a type of mastoid process which has been termed diploetic and which on section reveals its spaces to be filled with bone marrow. This is the typical embryonal type of mastoid process. Should the infection occur during the first stage of pneumatization we will have a partially pneumatized mastoid process, the pneumatization being limited to that area which has already been subjected to the ingrowth of connective tissue. The remainder of the mastoid process will be found to be spongy in character and to contain bone marrow between the bony trabeculae.

The development of the chronic suppurative otitis is in a large measure dependent upon whether or not a disturbed pneumatization has resulted from an infantile otitis. In the normal course of events, with a normally pneumatized mastoid process, an individual who develops a suppurative lesion in the ear will develop an acute mastoiditis rather than a chronic aural suppuration. In the normally pneumatized mastoid process the infection will spread by contiguity from the middle ear to the mastoid process and so result in an acute mastoiditis. In an individual wherein no pneumatization or a faulty pneumatization exists, the disease cannot spread into the mastoid process and is consequently limited to the middle ear, the antrum and the few neighboring cells which may have become pneumatized. This cannot conceivably produce what we commonly know as an acute coalescent mastoiditis. It may produce an acute otitis media, which will run its course and heal as such conditions usually do, or it may result in a chronic suppuration of one form or another, all depending upon the type of mucous membrane which has resulted from the otitis media neonatorum.

As we have said before, the type of mucous membrane remaining after the catarrhal type of otitis media neonatorum is a hyperplastic mucosa. It consists of a markedly hypertrophied subepithelial connective tissue covered by a high layer of epithelium. This high epithelium is not normal for the middle ear. It is due either to the replacement of destroyed areas by the epithelium from the mouth of the eustachian tube or to the effects of prolonged irritation by the foreign body in the middle ear. The hyperplastic mucosa also shows many adhesive bands in the middle ear between the drum and the ossicles and between the ossicles and the bony walls. These adhesions are the result of the hyperplastic outgrowth of the connective tissue in its attempt to encapsulate the foreign body. The hyperplastic type of mucosa, as we have stated, is always associated with a faulty pneumatization wherein the connective tissue grows into the marrow spaces but wherein the second and third stages of pneumatization are absent; in other words, with a sclerotic type of mastoid. It is in association with this phenomenon, primarily, that all forms of chronic otorrhea are found.

The chronic otorrheas are of two types, tympanic mucosal suppurations and suppurations kept up by the ingrowth of squamous epithelium. The former usually result from an ordinary acute suppurative otitis media. The tendency for an acute suppurative otitis to develop into a chronic purulent otitis media of the tympanic mucosal type depends upon the degree of hyperplastic mucosa present as a result of an infantile otitis of the catarrhal variety. This explains the finding of a sclerotic mastoid process in such cases. When we realize, from our clinical experience, that such cases are limited to the mucous membrane of the middle ear it is hard to understand, without Wittmaack's theory, why the mastoid process should suddenly become transformed from a pneumatized condition to complete bony density. We would have to show how a normally air containing cell, lined with epithelium, becomes obliterated by the formation of bone. This has, until now, not been demonstrated in any anatomic studies. Neither is it conceivable that bone will proliferate or be formed to such an extent in a space that contains air. If the bony trabeculæ did proliferate and increase in thickness, it would only be reasonable to expect that some evidence of remaining epithelial structure would be present in sclerotic mastoid processes; and this is not the case.

When, on the other hand, we understand that these chronic suppurations have as their predisposing and underlying factor a faulty type of pneumatization due to a previous infantile otitis, the finding of a sclerotic mastoid process in cases of chronic suppuration of the tympanic mucosa becomes clear. We realize that the sclerosis of the mastoid is not due to the chronic suppuration of the tympanic mucosa but precedes its advent and, in a way, predisposes to its occurrence. There are, of course, other factors which enter into the production of this type of chronic otorrhea. The chief of these is a chronic nasopharyngeal suppuration which results in repeated secondary infections of the middle ear.

Clinically the chronic purulent otitis media of the tympanic mucosal type of case is always associated with a large, persistent, central perforation which varies in size. The large perforation that we see in the fully developed case develops from a small perforation which was present during the acute stage of the lesion. The size of the perforation and its persistence are dependent upon the ingrowth of the squamous epithelium from the external surface of the drum. The squamous epithelium grows into the perforation until it reaches the mucosa of the middle ear. The mucosal epithelium in the center of the drum is anatomically lower than that found at the periphery. For this reason it does not resist the ingrowth of the squamous epithelium as well as the higher mucosal epithelium around the periphery. With the cessation of the acute inflammation scarification naturally occurs and there is a contraction of the intermediate fibrous layer of the drum. This causes the edges of the perforation to become more widely separated; and the separation continues until the margin of the squamous epithelium meets the epithelium of the mucosal layer at the free border of the perforation. With the approach toward the periphery of the drum, the ingrowth of squamous epithelium is stopped by the higher celled mucosal epithelium found in this vicinity. All further development in the size and shape of the perforation stops as soon as this occurs. Therefore, in a case of very marked hyperplasia of the mucosa, the perforation will probably be smaller than where the hyperplasia is not so marked. It will thus be seen that in this type of case the squamous epithelium plays its rôle, first, in the production of a persistent perforation, and secondly, in the size and ultimate shape of that perforation.

The second type of chronic otorrhea is dependent upon the ingrowth of squamous epithelium into the middle ear and its adjoining spaces. The different pathologic pictures which result are dependent, here also, upon factors which preceded the onset of the chronic otorrhea. The ordinary type usually develops from the same sort of lesion as precedes the chronic suppuration of the tympanic mucosa. It, too, is associated with a severe type of pathologic pneumatization due to a catarrhal infantile otitis. As we have seen, this severe type of infantile otitis media catarrhalis is accompanied by the development of adhesive bands in the middle ear. The determination of whether such a lesion will develop into a tympanic mucosal suppuration or a real chronic otorrhea due to the ingrowth of squamous epithelium depends directly upon the presence of adhesive bands at the margin of the perforation. When these are present, they afford easy access for the ingrowth of squamous epithelium into the middle ear. Macroscopically we find such cases exhibiting a marginal perforation. Where the epidermis grows into the middle ear, it can be seen otoscopically as a gray-white tissue, contrasting sharply with the red and inflamed middle ear mucosa. Where the two meet, there can usually be seen excrescences which take the form of granulations. These changes, of course, are very slow in their development. Microscopically we note that the squamous epithelium does not grow on to, and cover over, the mucosa of the middle ear, but definitely replaces it, so that as the squamous epithelium progresses in its growth into the middle ear it gradually destroys the mucosal covering. This explains the duration of these lesions over a great number of years. As long as the process of ingrowth of squamous epithelium continues, a chronic otorrhea will be present. This is due, not to a bone necrosis, as is commonly believed, but to the irritating effects of one type of epithelium upon the other. The squamous epithelium undergoes its normal process of desquamation, and the products of this desquamation act as foreign body irritants on the mucosa remaining in the middle ear. This produces a chronic inflammation of the mucosa which, in turn, reacts upon the squamous epithelium so as to produce a lesion of this structure not unlike a chronic eczema. This explains why, in the ordinary tympanic mucosal type of suppuration, we may expect a cessation of the discharge when the squamous and the mucosal epithelium meet at the border of the perforation so that the two layers of the epithelium no longer grow against

each other. The exudate of the one and the desquamation of the other do not come in contact with an epithelial surface to which they are foreign. Sometimes the mucosal epithelium is so high, as the result of an infantile otitis, that the ingrowth of epidermis is halted and the process comes to a standstill. This occurs mostly in the vicinity of the mouth of the eustachian tube.

Usually, however, the ingrowth of squamous epithelium continues until the entire tympanic cavity is covered with it. Where the antrum and aditus are completely filled with a strong hyperplastic tissue which has resulted from the infantile type of catarrhal otitis, the process then comes to a standstill and the lesion heals. Here we see something which is not uncommon in our clinical experience—a middle ear which appears uniformly white and wherein no evidence of mucosa is to be found. Such ears are dry; the promontory appears shiny; a large marginal perforation is evident; there are no evidences of repeated infections; the contour of the bony walls is maintained; and the major portion of the ossicles are seen to be preserved. We pronounce such cases to be healed cases of bone necrosis, basing our opinion on the finding of a marginal perforation and a sclerotic mastoid process. But these cases in reality are not cases of bone necrosis. An ingrowth of healthy squamous epithelium has replaced the diseased mucosal epithelium; and the compact mastoid process is the result, not of the recent suppurative lesion of the middle ear, but of a catarrhal type of infantile otitis with resultant faulty pneumatization. Such cases, when seen during their active stage, are usually interpreted as chronic otorrheas due to bone necrosis, and radical operations are performed for their cure. What nature is attempting to do, however, is to cure the lesion by replacing one type of epithelium with another, and in the performance of our radical operation we do the same thing: we merely replace the mucosal epithelium with a squamous epithelium derived from a plastic operation on the external auditory canal or from the grafting of skin.

In cases of strong hyperplastic mucosa wherein the antrum and the aditus have not been obliterated, the ingrowth of squamous epithelium is not limited to the middle ear but continues into these other spaces either by way of connective tissue bands or directly from the upper margin of the perforation. In such instances the desquamation of the squamous epithelium takes place

within the antrum, but the products of desquamation find their way out through the drum perforation.

Mass retention of desquamated epithelium never occurs in these cases, so that cholesteatome never results. This is due to the fact that the hyperplastic type of epithelium, high in character, exhibits a marked inflammatory reaction, as before noted. Consequently, the epithelial débris is thrown off and never accumulates. Such cases always heal without operative intervention.

The squamous epithelium also plays a rôle in the production of cholesteatome. The production of a cholesteatoma differs from the production of the ordinary type of chronic otorrhea in that here the middle ear and its accessory spaces are completely filled with the products of epithelial desquamation. These are not thrown off, as is the case in the type of chronic suppuration already discussed, but accumulate and cause a definite increase in the size of the spaces, with the characteristic clinical picture of a cholesteatoma. The mastoid process is sclerotic here also; but the sclerosis is not the result of the chronic otorrhea but is the preceding factor responsible for its cause.

Cholesteatomata are of two types, primary and secondary. The former is differentiated from the latter by the fact that no inflammatory lesion precedes its development. In this way it is distinguished from the ordinary chronic otorrhea described above. Clinically it is recognized when its full pathologic picture is present.

The primary cholesteatoma is developed only where there is a very marked degree of hyperplastic mucosa with mastoid eburnation following the catarrhal type of otitis media neonatorum. Consequently the sclerosis of the mastoid process precedes and is not the result of a cholesteatoma. When we realize that the marked sclerosis found in connection with a cholesteatoma is not the result of a new pathologic process but is the direct result of a faulty development in pneumatization we immediately see why it has never been possible to demonstrate, anatomically, the steps in the gradual transformation of a normally pneumatized mastoid process into a completely eburnized one.

In addition to the foregoing etiologic factors, the adhesive bands which are present in the middle ear may completely separate the aditus and antrum from the tympanum. In such a small

space as the tympanic antrum, the relatively thin area of the drum which we designate Shrapnell's membrane is thereby subjected to the negative pressure resulting from the resorption of air in the walled-off antrum and aditus. Such a case, in early life, presents the physical factors which are commonly associated with a vacuum. Air is absorbed. A transudation of serum occurs and the negative pressure causes a sinking in of this thin area of the drum. This results in the intrusion of a blind sac which intrudes into the recessus antri. Bezold[3] was the first to describe this. The blind sac which intrudes into the antral space is soon fixed to the walls of this cavity. Due to the markedly increased blood supply produced by the negative pressure on this area of the drum, a thickening of the cutis results. If the pressure is very great, the blind sac perforates. Then there occurs a real suppurative otitis, chronic in its pathology. The transudate present in the antrum cavity leaks out, and the squamous epithelium will grow onto the walls of the cavity, replacing the mucosa which is normally present in this area. The changes which then occur closely resemble those described when the products of desquamation act upon the mucosa and the mucosal secretion acts upon the squamous epithelium. Granulations are present in the area between the mucosal epithelium and the squamous epithelium. On otoscopic examination a foul, seropurulent discharge is present which is seen coming through a perforation in Shrapnell's membrane. The remainder of the drum is seen to be intact. In addition one finds a collection of desquamated epithelium in the region of the perforation. The aditus and the antrum are gradually converted into a smooth cavity, lined with squamous epithelium. This cavity gradually enlarges, due to the increase in the amount of the retained desquamated epithelium. In contradistinction to what occurs in the ordinary type of chronic otorrhea, the cavity becomes filled with desquamated epithelium and soon assumes a tumorlike form. The growth of this tumor is dependent partly on mechanical factors and partly on biologic factors.

Mechanically, we must consider the difference in size between the small perforation which results from the negative pressure exerted on the blind sac and the cubic capacity of the space formed by the antrum and aditus. The relative smallness of the perforation as compared to the large capacity of the antrum space results in the mechanical retention of desquamated epithelium which by its own pressure causes an increase in the growth of

its mass. This, however, does not in itself explain the growth in size. Despite the fact that Erdheim, Linck[4] and Grünwald[5] give this as the principal cause, certain biologic factors must also be brought into consideration. Manasse[6] was the first to explain them. They consist, in the first place, of an increased activity in the proliferation of the squamous epithelium with a concomitant destruction of the mucosal epithelium, and, in the second place, of the difference in the type of subepithelial connective tissue encountered by the squamous epithelium.

The greater proliferation of the squamous epithelium is due to the fact that in the antrum and attic there remains a relatively low epithelium. When we consider that production of a primary cholesteatoma is dependent upon the absolute shutting off of the antrum and the aditus from the remainder of the middle ear, we must realize that the high celled epithelium which proliferates from the tubal surface over the middle ear cannot possibly enter the antrum and aditus, since these spaces are completely separated from the remainder of the tympanic cavity by the adhesive bands resulting from an infantile catarrhal otitis media of the severest type. This is one factor which favors the rapid ingrowth of the squamous epithelium. In addition, the subepithelial connective tissue retains its loose, delicate and richly vascular qualities, which are not different from those found in the normal embryonal state. Consequently, it affords an excellent matrix for the squamous epithelium to grow over, for it produces excellent nutrition for this tissue which is destined to become the cholesteatomatous matrix.

The ingrowing squamous epithelium proliferates tremendously on this new vascular connective tissue upon which it is transplanted and it exhibits a marked growth, much greater than that found in the normal proliferation of squamous epithelium. It tends, therefore, to form itself into ridges or folds. However, since the configuration of the antrum does not permit this and since the retained products of desquamation exert a pressure upon the growth of the squamous epithelium, the latter can find room for its increased growth only by an eccentric spread into the underlying tissue. This spread must occur at the expense of the loose subepithelial tissue. This in turn causes an attempted regeneration of the normal subepithelial tissue, which can take place from one source only, since there is no longer connective tissue present to replace that destroyed by the growing cholestea-

tome tumor. The only connective tissue from which regeneration is possible is the perivascular connective tissue present around the vessels in the compact bony structure which borders the antrum space. This perivascular tissue forms characteristic proliferating foci which consist of loosely vascular connective tissue and which develop into typical Howship's lacunæ. These eventually spread, proliferate and grow into the subepithelial tissue.

This process causes the bone which is enclosed between the resorption foci to be destroyed and absorbed; and the cholesteatomatous cavity increases in size at the expense of the underlying bone. The rapidity with which this cavity increases depends upon the vascularity of the adjacent bone. The more compact the bone and poorer in blood supply, the slower the growth. The less compact the bone and the more vascular its character, the more rapid will the growth of the tumor mass be.

The resorptive foci of connective tissue which are present in the bone are not to be looked upon as the results of an inflammatory process, as has been described by Haike,[7] Zange, Kutvirt and others. An inflammatory process, in order to produce such foci of resorption in the bone, must first produce a complete necrotic destruction of the overlying mucosa; and this is not present in primary cholesteatoma.

The tendency of a primary cholesteatomatous cavity to expand is lessened by an increase in the size of the perforation leading to the external canal. This opening may become larger through destruction of the outer aditus wall or of the posterior bony canal wall, in which instances the cholesteatomatous cavity is brought into direct communication with the external auditory canal. In addition, any secondary infection which may occur in the cholesteatomatous cavity will decrease its tendency toward growth, since the invading organism will produce a suppuration with a discharge of cholesteatomatous material into the external auditory canal. The primary cholesteatoma are usually discovered late in life. The patient almost invariably denies knowledge of any suppuration in the middle ear. The clinician wonders at the fact that he finds an attic perforation on clinical examination and discloses a large cholesteatomatous cavity at operation, and yet the patient has never known of a suppuration of the middle ear. Such cases have their onset in a marked hyperplastic mucosa with faulty pneumatization, which results in a marked hyperplastic mucosa and sclerotic mastoid process in later life. An

infection has never been present, and the product of the cholesteatoma is dependent, not upon the ingrowth of squamous epithelium secondary to an inflammation of the middle ear, but upon mechanical factors in infancy, which have separated the antrum from the middle ear. These mechanical factors at first produce a marked retraction with subsequent rupture of Shrapnell's membrane. The epithelium, encountering a rich vascular matrix, rapidly grows, and the narrow perforation which results is not sufficient to discharge the products of desquamation. The mechanical factors of pressure then cause a destruction of this vascular matrix and a replacement of the connective tissue from the vascular spaces of the surrounding compact bone.

There is another form of chronic purulent otitis media which is associated with the anatomic conditions of severe hyperplastic mucosa of the middle ear and a sclerotic mastoid process due to the catarrhal type of otitis media neonatorum. This type of otitis usually occurs in scarlet fever and in connection with debilitating diseases like diabetes or nephritis. In brief, it is produced by a widespread thrombosis of the vessels of the submucosa, which leads to a mass necrosis of the mucous membrane. In very severe cases this tissue necrosis involves the periosteum of the middle ear and antrum. In mild cases, where the necrosis is limited to certain isolated areas, complete healing may occur by a proliferation of the remaining mucosa and a covering over of the denuded areas. In extensive necrosis the bone becomes involved, not by the infection but in the process of healing. This process is known as rarefying osteitis or bone caries and is the result of proliferation of the connective tissues surrounding the vascular spaces in the bones. This connective tissue grows into the middle ear cavity and in its growth surrounds areas of bone which are later thrown off as sequestra. As an end result, the entire drum is destroyed and the middle ear is filled with connective tissue granulations which arise primarily from the resorptive bone foci. The healing of this lesion is left to the squamous epithelium of the external auditory canal. This begins to grow into the middle ear at the margins of the annulus. As the process goes on, the originally delicate and vascular connective tissue which arises from the vascular channels in the bone is gradually changed into a more or less fibrotic mass which fills the entire middle ear. This appears clinically as granulations. At the depths of this layer of granulations new bone formation occurs which can be demonstrated

microscopically. In this way, little by little, a distinct boundary between the bony walls and the soft connective tissue granulations is obliterated. The entire antrum may become obliterated by the new bone formation, and there may eventually occur a complete scarification of the tympanic cavity due to the contraction and ossification of the granulations which are covered by squamous epithelium. If the antral lumen is retained as such, the squamous epithelium grows into this, and the necrotic otitis becomes a chronic purulent otitis media due to the formation of a secondary cholesteatoma. The development of a secondary cholesteatoma is dependent upon the presence of a wide antrum in a case of acute necrotic otitis. A secondary cholesteatoma cannot develop where the antrum has been obliterated or where a good cellular development of the mastoid is present, since a preformed space is absent in the first instance and an acute mastoiditis usually develops in the second. In other words, a preformed space must be present in a case of chronic otitis due to bone caries, as it is commonly known, in order for a secondary cholesteatoma to develop. This explains why secondary cholesteatomatous formations may occur following a simple mastoid operation which creates a large space into which the squamous epithelium of the external ear may grow.

The reason for the ingrowth of squamous epithelium is the fact that in cases of chronic otitis large areas of mucosal epithelium have been destroyed, and their replacement must take place either from the eustachian tube or from the external auditory canal. Since a necrotic otitis is an ascending infection from the pharynx and spreads by contiguity, the tubal epithelium is usually destroyed and the tube closed by adhesions. The marginal perforation which is present in such cases favors the ingrowth of squamous epithelium from the external auditory canal. This ingrowth proceeds far more quickly than in the ordinary type of chronic purulent otitis media because there is no mucosal epithelium to be destroyed before the squamous epithelium can find an area to grow over the bone. It grows rapidly over the granulation tissue which is the loose and very vascular tissue remaining after the necrotic bone has been thrown off. The secondary type of cholesteatoma attains a large size very quickly because there is a large preformed space in the antrum. The subsequent growth of the cholesteatoma proceeds exactly as described above for the primary cholesteatoma. Macroscopically the drum is totally de-

stroyed and a large defect is noted. The ossicles are also seen to have been destroyed in the process of necrosis. The walls of the middle ear are gray-white in appearance, due to their lining of epidermis. Cholesteatomatous masses are seen to come away from the region of the antrum and attic. Clinically it is almost impossible to distinguish between the primary and the secondary cholesteatomata in their late stages. The developmental picture described here is seen early.

The foregoing shows us that all chronic suppurative lesions of the middle ear, wherein a marginal perforation exists, show an ingrowth of squamous epithelium in the ordinary type of chronic purulent otitis media. This ingrowth causes a healing of the lesion to take place. In the necrotic otitis, which results in a chronic otorrhea, the squamous epithelium endeavors to heal the lesion; and, if the antrum is obliterated by scarification, healing actually takes place. In the case of the necrotic otitis, with a large preformed antral space, the attempted healing of the lesion by the squamous epithelium is frustrated by retention of desquamated epithelium. We therefore see that nature attempts to heal chronic suppurative lesions of the ear by the replacement of mucosal epithelium with squamous epithelium. It is only where insufficient drainage is present that nature is unable to do this. We utilize this fact unconsciously in the performance of the radical mastoid operation. We create a large cavity which we line with squamous epithelium derived from the external auditory canal or from a skin graft. We permit this epithelium to grow over a bone surface which we have denuded of all mucosal epithelium. In other words, we have created a healed chronic purulent otitis media and have forestalled its further development by permitting adequate drainage so that none of the products of desquamation can be retained in our artificially produced cavity.

Since this is the case, we must study more carefully the type of chronic otorrhea which presents itself to us. All cases presenting a marginal perforation are to be viewed as cases of chronic purulent otitis media which will eventually undergo a transformation of the mucosal epithelium of the middle ear into a covering of squamous epithelium derived from the external auditory canal. It is only the primary type of cholesteatoma, produced by an ingrowth of squamous epithelium due to mechanical factors, that is really not a chronic suppuration but rather a tumor-like formation. Such cases require the removal of the

tumor by a radical mastoid operation. The remaining cases are cases of true chronic otorrhea which are being healed by the ingrowth of squamous epithelium. Such cases should, after a period of years, be completely dry if nature is successful in its replacement of one tissue by another. The unsuccessful attempts are due, first to poor drainage, so that a secondary cholesteatoma forms which must be removed, and second, to a continued rarefying osteitis which constantly throws off sequestrated bone and consequently does not permit the epidermis to cover it. The latter type of cases are those wherein the new connective tissue never undergoes fibrosis and consequently no scarification results. Such cases require, not a radical mastoid operation, but merely a removal of the diseased bone so that the epidermis can grow in and cover over the area.

Another factor to be borne in mind, and one of greatest importance, is that sclerosis of the mastoid process is not the result of a chronic suppuration but precedes it. In itself it is of no diagnostic nor prognostic value, and it serves no guide as to the therapy indicated. We should judge our cases clinically by the appearance of the drum and of the inner tympanic wall, by the presence of an antral space and by the character of the discharge. The perforation in the drum determines whether the case is one of tympanic mucosal suppuration or whether it is one wherein the squamous epithelium will play a role in its further development. The color of the inner tympanic wall will show whether or not nature is successfully healing the lesion. Where it is smooth and shiny and gray-white in appearance over its entire extent, we can be certain that the squamous epithelium has completely covered and healed this area so that in itself it will no longer be a factor in the production of a chronic discharge. Where the walls show granulations we can feel that the underlying lesion is still active. When such a picture is seen after a chronic otorrhea has been present for a number of years, it speaks for an underlying rarefying osteitis with sequestra. A large attic and antrum, determined by a probe inserted into these spaces, should always suggest the possibility of a cholesteatoma developing secondary to the attempted healing of the lesion by the ingrowth of squamous epithelium. Lastly, a discharge containing dry, flaky desquamated epithelium indicates that the squamous epithelium has already invaded a preformed antrum space and will produce a growing cholesteatoma.

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DISCUSSION.

Dr. Samuel J. Kopetzky, New York City: When one has travelled along a road rather rapidly and has failed to note the signposts, he is apt to be annoyed when he enters a cul de sac and is obliged to retrace his steps until he reaches the signpost that points the way. That is exactly what has happened to otological pathology—going forward fast and failing to note the monument erected by Wittmaack in 1918. We who have travelled beyond the signpost must now carefully and painfully retrace our steps to 1918, to an understanding of the work of Wittmaack, an understanding of the pathological entities with which we deal. I recommend the study, the careful re-study and re-analysis of Wittmaack as the basis of the work that Dr. Almour has presented this afternoon, because this study of the epithelium explains much that heretofore we have not been able to understand. It permits a comprehension of pathological pictures as we find them on the operating table. It not only does that, it often determines the position and location of the perforation and almost automatically classifies the case. It determines a thing which I have clinically asked you to accept for some years, namely, that we cannot say that the acute case is of short duration and the chronic case of longer duration, but that chronicity of certain ear lesion is an entity from the start. Furthermore, a revaluation, or rather a revisioning in the reading of our Roentgen plates is necessary. Everything that does not show cell trabeculae should not be read as sclerosis, and the Roentgenologist must differentiate between cell destruction, fibrosis and sclerosis. These pictures then studied in the onset of infection will at once classify your case, because from what you have seen this afternoon, it is impossible to have an acute condition under certain pathological situations.

It is interesting to go over the careful reports of Dr. Beck, who reported on cases and then had a chance to see them again after fifteen

years. He shows not sclerosis, but fibrosis. He is showing additional contributory evidence of the condition presented this afternoon.

In all the hundreds of laboratories and places where careful workers have studied there has never been a report of the new growth which would be necessary in the development of sclerosis as a factor other than as developed due to faulty pneumatization caused by embryonal and infantile ear disease. Specimens should be at hand showing the transition from the pneumatized to the nonpneumatized structure, whereas we have specimens of the pneumatized in various degrees of reaction to bacterial infection. We have specimens of the fibrotic replacement and pneumatization, and we have specimens of sclerosis as we find it when we do a radical operation, without quite understanding the reason for the lesion for which we are operating.

Finally, if we carefully read the paper Dr. Almour has presented this afternoon we will find the key to the logical application of therapeutic agents and we will not apply agents which will not influence the pathology presented by the lesion.

Dr. E. M. Seydell, Wichita, Kansas: The studies of Wittmaack have given us a satisfactory explanation of many of the variations which occur in the course of inflammations in the middle ear and mastoid. They help us in making a differential diagnosis and prognosis and are of immense value in determining the method of treatment required for the individual case.

In a study of the literature on this subject one finds that some of the Wittmaack principles have not been generally accepted. Albrecht has come to the conclusion that infantile infections are not the only cause for variations in pneumatization. In fact, he thinks the fundamental factor underlying these variations is the individual cell potentiality. In order to prove this theory Albrecht made a Roentgen ray study of the mastoids of 60 identical twins and of 50 fraternal twins. In the identical twins the degree of pneumatization corresponded in the four mastoids in 66 per cent., whereas in the fraternal twins this similarity in structure occurred only in 37 per cent. Further, in the mastoids which were the best pneumatized the structure was uniform in both sets of twins in 92 per cent. Where the pneumatization was less perfect, this percentage decreased to 74 per cent, and where the pneumatization was only moderately developed the similarity occurred in only 42 per cent. In the compact form the percentage increased to 66 per cent. From these findings he drew the following conclusions:

1. A healthy mucous membrane may develop in spite of external influences.
2. Where a diminished individual cell energy is present, external influence increase in importance, i.e., infection, etc.
3. In the compact form of mastoid the mucous membrane which was incapable of producing pneumatization resembled the membrane having the normal amount of cell energy in so much that it could not be influenced by external agencies.

Following the study he made a histological examination of the mastoids of 44 newborn infants to determine the variations in the mucous

membrane and the factors causing them. His findings agreed with those of Wittmaack. A bilateral similarity in the structure of the middle ear and mastoid was found in 39 of the 44 cases; there was no relationship between the degree or type of pneumatization and the degree of infection found in the middle ear. He confirmed the previous findings of Voss, the presence of numerous hemorrhages in the mucous membrane of the middle ear, and thinks this is one of the causative factors which prevent the absorption of the embryonal connective tissue.

Mouret and Portmand disagree with the infection theory of Wittmaack and agree with Albrecht. They point out that if Wittmaack's theory is true we would have more compact mastoids, since Wittmaack claims infectious in the middle ear in 90 per cent of the newborn.

The statement that it has not been shown by anatomical studies where or how a normal pneumatic mastoid may develop a complete bony density I do not believe is generally accepted. N. Pauton, in an article on the histology of mastoiditis, reports the histological examination of bone removed from 65 mastoids which had been operated on account of mastoiditis, and in 45 of these cases there was marked new bone formation. At the end of the third week the pneumatic cells may be filled with fibrous granulation tissue and the epithelial lining may be entirely destroyed. At this time the picture of inflammation in a well pneumatized mastoid resembles that of a primary spongy mastoid. Sclerosis of the mastoid is the result of the spontaneous healing following infection.

The statement that cholesteatoma develops only in sclerotic mastoids has been disproven by Ruttin, O. Beck and Urbantschitsch. Ruttin and O. Beck have each reported two cases in which a cholesteatoma was found in the attic of a patient suffering with an acute mastoiditis. In all of these cases the patient had a pneumatic mastoid. The cholesteatoma had been present for a long time. Urbanschitsch reported three cases in patients whose mastoids also were pneumatic.

Dr. S. J. Pearlman, Chicago: The orator is to be congratulated on his simple and careful exposition of a topic which, as time goes on, will be more and more discussed and found important. A good many years ago it was thought that the changes that took place in chronic otitis media were due to hyperplasia of the lining of the middle ear; later it was proven that it was the epithelium from the external auditory canal that was responsible for the changes that took place. It must be remembered, however, that this ingrowth of epithelium in the middle ear is primarily an attempt by Nature at healing. If in this attempt cholesteatoma is formed, that is an unfortunate consequence.

Dr. Kopetsky called attention to some changes of opinion that have occurred, but the main change of opinion to my mind is in regard to the importance of bacteriological studies in chronic otitis and acute otitis. It would seem the tide is swinging away a little from these and less importance attached to the type of bacterial invader.

It becomes evident that it is necessary also to study the changes that are taking place in the tissues of the middle ear and mastoid due to infection or trauma in early life. At one time it was thought by some that if in the culture hemolytic streptococci were found it was almost sufficient to warrant surgical interference. We know now that by careful study we

may be able to say very early that this ear is to be a chronically infected ear, or that ear is the type in which there will be a coalescent mastoid, without paying so much attention to the bacteriological findings.

A point that Dr. Almour did not mention—it is not a part of his topic—is that more and more attention is being given to this attempt by Nature at healing by invasion of the epithelium in carcinoma. It becomes plain that the cause of chronic otitis is often due to epithelization by way of the external auditory canal. Tissues will fight such invasion under some circumstances; under others they will not. The vigor of the resistance varies, depending partly on constitutional factors as yet not understood. In the instance where the ingrowing epithelium meets much resistance instead of spreading out and possibly producing healing it is compelled to dig down, and in the entrenchment process the deeper layers of the epithelium assume the aspects of carcinoma. Only recently I noticed an interesting report in which there was demonstrated by very unusual and clear illustrations this downward digging of the epithelium and the very earliest appearance of carcinoma of the squamous cell type.

Dr. Joseph C. Beck, Chicago: I wish that my chief, Dr. Norval Pierce, could be here to hear this discussion. He has been such an ardent admirer of Wittmaack—all who have read Wittmaack's monograph admire him—and this discussion is so refreshing and hopeful for a better understanding of the pathology of the mastoid. It has been my work and pleasure to study microscopic sections of the mastoid tips in many cases we have operated, both acute and chronic, trying to fit this into Wittmaack's work, but I must say there are many variant and opposite opinions, and in most cases of the type that Dr. Seydell quoted. That is one man's work, and it will take a great many studies to prove the facts. If they can be proven in many cases I think modern otology will be rewritten on the basis of Wittmaack's conclusions, although there are many opponents in Europe because there is a great deal of animosity in scientific work there.

My case that Dr. Kopetzky quoted was that of a medical student. She went as a missionary to a foreign country and remained for a number of years. I saw her again when she was suffering with otitis media and took X-ray pictures of both mastoids, and they were well pneumatized. She came back a year later with labyrinth symptoms and cholesteatoma present. I operated radically and studied the specimens and found there was fibrosis—a different classification of the change in the bone. Fibrosis and sclerosis are two different things so far as this particular anatomy is concerned. I like to study histologically and anatomically conditions referable to the mastoid and apply it to the diseases we are accustomed to study.

Dr. Bernard J. McMahon, St. Louis: I was very much interested in Dr. Almour's exposition and explanation of the pathologic changes in the middle ear, particularly because our recent findings do not seem to corroborate all of them. There are two or three points on which I would like to express an opinion, if for no other reason than to raise other controversial points for my own information as well as for that of others.

The Doctor mentioned the middle ear's being normally lined with cuboidal epithelium, that is, first the middle ear contains mesenchyme, this is absorbed, then it is lined with cuboidal epithelium. Our findings so far

suggest that the middle ear is originally partially lined with stratified columnar ciliated epithelium similar to that lining the eustachian tube. After infection has existed for some time, this appears to become transitional or cuboidal, and the cilia are lost. The antrum and para-antral cells and the bone spaces while originally filled with mesenchyme, have been found to be lined with cuboidal epithelium on the surface of the contracted tunica propria, or adult mesodermal tissue. That is one guide we have used in distinguishing bone spaces, from normal middle ear cavity and marrow spaces. The marrow spaces are entirely different. They seem to be connected in the periphery with the para-antral cells by the tunica propria only, transmitting blood capillaries, and have no lining epithelium.

Another point that I cannot understand is, how the mesodermal tissue can be resorbed and then be replaced on the surface with epithelium that is endodermal, unless this endodermal tissue was originally there. There is no transition from one embryological layer to the other.

We have found fibrosis in the mastoid of an infant as young as three weeks of age, with a period of clinical symptoms as short as eight days. Of twenty-five infants ranging in ages from three weeks to thirteen and a half months, sixteen showed a preoperative infectious period of less than four weeks, seven of these being less than ten days. Consequently it seems to me that these fibrotic changes are the result of an individual proclivity to the formation of fibrous tissue rather than a developmental or an infectious factor. In our work the infants having mastoids of the fibrotic type showed a greater percentage of recoveries than those of the oedematous type.

Another observation we have made is that in fibrosis of the mastoid in certain adults, in the course of an infection, there seemed to be taking place a resorption of calcium, the bone cells remaining alive and resuming their activities as connective tissue cells. I think that is one explanation for the sclerotic mastoid. The embryonal tissue we believe persists throughout life, not as embryonal tissue in the strict sense of the word; but as the normal tunica propria. If it becomes oedematous in response to infection, it assumes its original appearance of embryonal tissue and after infection subsides it resumes its normal adult character. Fibrosis takes place in proportion to the number of connective tissue cells that remain after infection. The presence of young connective tissue cells does not mean that fibrosis must ensue, the connective tissue cells are young and can migrate to other parts, leaving the invaded tissue in its pre-infected condition.

DR. RALPH ALMOUR, closing: I want to thank the discussors for their reception of the paper. I think Dr. Seydell has about summed up all the principles against the Wittmaack theory. A great deal has been written about it and we have only taken up a few points in it. As regards Albrecht's work, in his radiographic work he comes to conclusions that do not corroborate Wittmaack's. On the other hand, his histologic studies agree with Wittmaack. Wittmaack's work is based entirely on histologic findings.

Dr. Kopetzky and I have repeatedly X-rayed the cases that come in regardless of whether they have ear complaints or not to determine the status of the mastoid process. We have cases wherein there was no history whatever, from parents or the patients themselves, of any kind of ear disease, and yet we have found real sclerosis of the mastoid. On the other hand, we have cases wherein some ear pains were present, no suppuration at any time, patients in which the adenoid had been removed, and these patients at ten or eleven showed no pneumatization of the mastoid process, and yet no suppuration had ever been present. I just state this as a fact.

As regards perfect pneumatization being present with infection, that is possible if otitis media of the suppurative type happens to come on after the first stage of pneumatization has been completed. In other words, if the invasion of the bone spaces with subepithelium has taken place, the ingrowing epithelium covers these spaces already pneumatized, and infection occurring early in life, after the child is six or seven months old, will have absolutely no effect on that portion that is already pneumatized.

As to hemorrhage from the middle ear previous to pneumatization, that is possible because it is a foreign body. The question of cholesteatoma which would be present in a non-pneumatized mastoid is a fact that has been reported. The explanation is that probably there was an abnormal patency into which the epithelium probably grew.

Dr. McMahon's findings of columnar epithelium is not new; all sorts of epithelium have been reported, but from all the work on that subject it is very reasonable to assume that these findings are not normal findings. In other words, the normal mucosa of the middle ear is lined by cuboidal epithelium and any destruction of this epithelium will cause it to be replaced by epithelium like that of the eustachian tube. We may find areas of lymphoid tissue in the middle ear, but that is not normal, it is the result of an old pathologic process. My findings on my slides will show a layer of cuboidal epithelium. We have seen others that agree with Wittmaack, but that is not the normal type for the histologically normal middle ear. The finding of cuboidal epithelium is perfectly normal because that is exactly what grows from the middle ear into these cells.

I want to make one more point, that we unconsciously have been using squamous epithelium to line the middle ear. We take squamous epithelium and put it into the cavity after we have removed the mucosa. Nature is attempting to do it all the time, and I believe if we can determine clinically whether or not the squamous epithelium which is growing in will not enter the antrum, which is present as a space, we can safely permit the epithelium to grow. If, on the other hand, the antrum is present, some kind of surgery must be performed, not necessarily radical, so as to create a large space wherein no epithelium debris will collect and produce tumor formation.

CHRONIC DIPHTHERIA ORIGINATING IN AND LIMITED TO THE TRACHEA AND BRONCHI.

ALBERT E. BULSON, M. D., AND EUGENE L. BULSON, M. D.,

FORT WAYNE, INDIANA.

Diphtheritic infection primary to the trachea and bronchi is of relatively uncommon occurrence, though by no means unknown. Diphtheria, chronic in type, is also recognized occasionally, though this, too, must be regarded as unusual. Chronic diphtheria originating in the trachea and limited to that region throughout its course must be regarded as an exceedingly rare condition. No case exactly paralleling the one to be described has appeared in a search covering the literature for the past fifteen years.

In a monograph *Ueber Bronchialdiphtheria*,[1] published by Max Reh, in 1912, several cases are quoted from an article by Hennig, entitled *Ueber chronische Diphtheria*, that have some points of resemblance. In one a three-year-old child had exhibited for two weeks a slight dyspnea with some cough, no reason for which could be found in the pharynx, larynx or trachea. After a time diphtheria appeared in the larynx and tonsils, and in the tracheobronchial system as well. Unfortunately no lung findings had been recorded. Henoch also reported a case of a child who had been intubated, who coughed up a membrane from the deep lung passages fifteen weeks after the removal of the cannula. Reh also relates a case, which came under his own observation, of a young man who entered the Greifwalder Clinic with a tentative diagnosis of pulmonary tuberculosis. The sputum was negative for tubercle bacilli but positive for the diphtheria organism. At the original examination no suspicion of diphtheria was entertained. A week later, however, diphtheria of the pharynx made its appearance, and after this had cleared up under treatment tubercle bacilli were found in the sputum.

Lynah[2] made a special study of tracheobronchial diphtheria and reported a considerable number of cases, seen for the most part at the Willard Parker Hospital, New York. In 1916, he reported thirty-eight cases, and in two later reports added several further observations. As the Willard Parker Hospital is maintained for

cases of contagious disease only and is free, the cases encountered there are likely to be severe in type and the patients seen only when the condition is acute. Lynah says: "The clinical picture of a case of tracheobronchial diphtheria is quite distinctive of this disease. The onset seldom is sudden and the voice never is lost, and aside from an occasional cough and slight stridor, the condition may be readily overlooked and treated as an ordinary catarrhal cold. . . . There is no doubt that many are overlooked, unless a case of membrane is coughed up, or the larynx becomes involved with a typical picture of laryngeal 'croup.' It is only then that a correct diagnosis is made. There is no doubt that many of the so-called asthmatic attacks which terminate fatally are due to diphtheritic bronchial obstruction. . . . . Any slowly progressive dyspnea should be looked upon with a considerable degree of apprehension, and when we are unable to account for the cause of the dyspnea, a bronchoscopic examination should be made. . . . In tracheobronchial diphtheria the process is a mechanical obstruction to respiration, plus carbonic acid poisoning and diphtheritic toxemia."

Lynah had seen several cases of tracheal diphtheria in private practice where the disease was early recognized. All these patients recovered after a dose of 10,000 units of antitoxin subcutaneously; there were parietal casts in all, but intubation was not necessary. "There is little or no prostration early in the disease, and postdiphtheritic paralysis is never seen, unless the case is of long standing and the pharynx and tonsils have become involved. Even enormous dosage of antitoxin and mechanical removal of the membrane in the case of long standing is futile, for these patients invariably succumb."

Lynah found it comparatively easy to remove the membrane in early cases, making use of a vacuum tube. The trachea and the bronchial system were then swabbed through the bronchoscope with antitoxin to prevent reformation of the membrane, and long tracheobronchial tubes were introduced to prevent stenosis in case the membrane should reform and fill with blood clot.

During the ten years which elapsed after Lynah made his publication a number of single cases of diphtheria of trachea or bronchi were reported,[3 4 5 6 7 8 9 10] but the clinical picture described was practically identical, and all the comments were merely "rehashed" from Lynah, many being transcribed verbatim. Carpenter,[10] of South Carolina, saw, in consultation with a coun-

try practitioner, a five-year-old child, who had been ill for five days with what was supposed to be the aftermath of influenza. When seen by Carpenter the fauces, larynx, trachea and bronchi were covered with a diphtheritic membrane, although the practitioner assured him that there had been no signs of any membrane at his last visit to the patient only a few hours before the consultation was held. Carpenter passed a bronchoscope and extracted membranous casts from far down the bronchi and the trachea, which were necrotic, showing that the casts had been in existence for several days at least. The dyspnea was relieved, but the child died of diphtheritic toxemia.

A cursory examination of reports of diphtheria "carriers," which are extremely numerous in the literature, has not shown anywhere that the organisms were confined to the trachea throughout, as in our case under consideration.

Case Report.—A housewife, aged 67 years, first consulted us September 29, 1928. She gave a history of having had influenza 10 years previously and general poor health since, but at no time was she confined to bed. She had no cough except occasionally when a membrane was coughed up without difficulty. This had been going on for several months. Usually this membrane was a very small piece, though occasionally it was a piece as large as the little finger nail. Sometimes no membrane was coughed up for a period of a month, and then again pieces were coughed up within a few days or a week of each other, and accompanied by little or no mucus. During the interval there was no cough, no phlegm or mucus in the throat, and no soreness or pain in either throat or chest. So far as the patient knew there had been no fever.

Upon examination the nose and throat appeared essentially normal, and a very careful laryngoscopic examination disclosed no abnormality in the larynx or trachea. As the patient appeared to be in fairly good health for a woman of her age, and was doing her own housework, she was advised to save some of the membrane coughed up and bring it for examination. Within a few days a small piece of dirty white membrane was brought in and submitted to a pathologist, who reported that both smear and culture showed diphtheria bacilli. The patient returned a few days later complaining of some discomfort in the windpipe, with a feeling of obstruction and slight disturbances in breathing. With the laryngoscopic mirror some of the membrane could be seen immediately beneath the vocal cords, and dur-

ing the examination this membrane was coughed up and proved to be about the size of a ten-cent piece. This specimen was submitted to the pathologist, who again reported that in both smear and culture diphtheria bacilli were found. The patient had no fever and no increase in pulse rate. Chest examination was negative except for a few rales. Nose and throat were absolutely clear.

Forty thousand units of antitoxin were administered, without incident, and a few days later small pieces of membrane and some thick mucus were coughed up, and the pathologist reported that they gave an almost pure culture of diphtheria bacilli. Again 40,000 units of antitoxin was administered, but this had no particular effect. A week or ten days later more diphtheritic membrane was coughed up and again the pathologist reported the presence of diphtheria bacilli. During this treatment the patient had been feeling very well, was ambulatory, and exhibited no rise of temperature or pulse rate.

Upon the suggestion of the pathologist, autogenous vaccine treatment was started. The vaccine treatment was begun on October 15th, a dose just short of reactional being given every five days and the doses gradually increased. The vaccine treatment was continued with only short intermissions until August, 1929, or for a period of approximately ten months, and during that time membrane was coughed up at least two to six times per month, and in every instance the pathologist reported almost pure diphtheria bacilli. As a check, specimens were submitted to a second pathologist, who likewise reported the infection as diphtheritic. Once during that period, or on November 5, 1928, the membrane assumed the form of a cast of the trachea and was about three-quarters of an inch in length. Five days later, or on November 10th, another smaller cast was coughed up. On August 27, 1929, the family physician decided to try toxin-antitoxin, and administered the regular course of toxin-antitoxin injections. This treatment seemed to be ineffective, so she returned to vaccine treatment, which was continued until November 23, 1929, when it was abandoned. Aside from the antitoxin, toxin-antitoxin and vaccine treatment, the patient had no other treatment except inhalations of compound tincture of benzoin vapor in connection with the inhalation of oily sprays.

The patient continues in fairly good general health, doing her own work, has no fever and has no cough except when cough is

required to expel some of the membrane which still accumulates and is expelled on an average of two or three times a month. At no time since first seeing the patient, or until January 1, 1930, over a period of fifteen months, has there been any membrane above the larynx, nor could we procure diphtheria bacilli from the smears or cultures from tissues above the larynx.

A bronchoscopic examination never was made, as the patient objected to that procedure, though we are of the opinion that local treatment through the bronchoscope offers the best chance of relieving the condition and we would have employed the method had the patient consented.

As already stated, the case is unique and unusual, if we may judge by the literature of the last fifteen years, both in this country and abroad, which has been searched very carefully.

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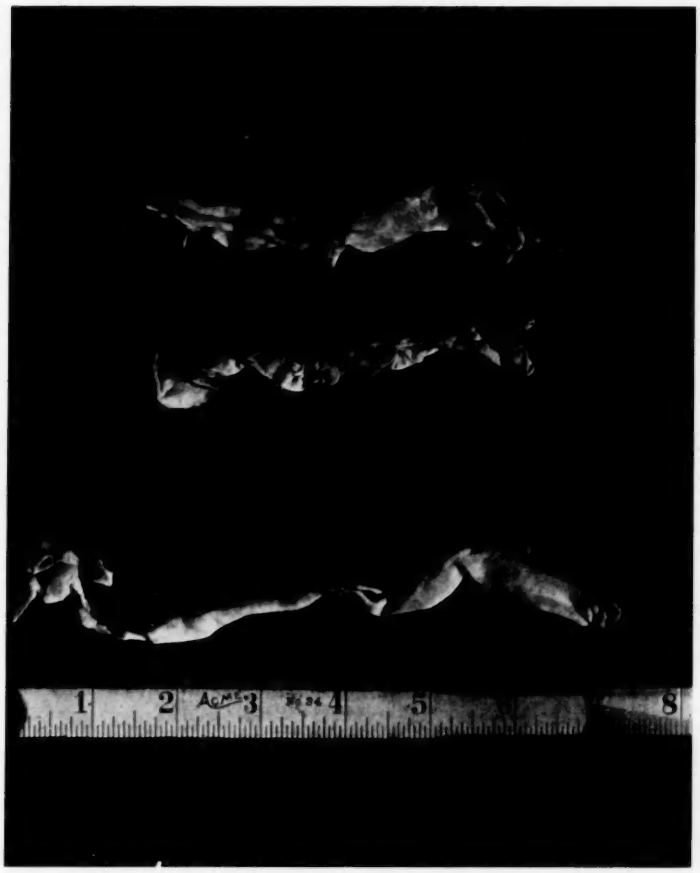
*This reference is wrongly given in the Surgeon-General's Index as *Trousseau*, with the volume number as LII. This mistake was copied by Boebinger in his bibliography.

DISCUSSION.

DR. GABRIEL TUCKER, Philadelphia: When I was asked to discuss the presentation of Doctors Bulson I hesitated to do so because I thought I had had no experience with chronic diphtheria of the trachea and bronchi; but after going over their excellent paper and case report I wonder if I have not been overlooking some of these cases. We have all seen a considerable number of cases of chronic tracheobronchitis where there was crusting of the secretions in the trachea and bronchi, with atrophic changes

in the mucous membrane. Frequently there is an atrophic rhinitis associated in these cases. In the bronchoscopic study of these cases routine bacteriologic studies were made and organisms of diphtheria were not reported. In the future we shall make more careful studies in this respect. Bronchoscopic examination will give the best opportunity for both diagnosis and local treatment of this condition.



Casts of tracheo-bronchial tree coughed up in tracheo-bronchial diphtheria in a boy 18 years of age.

I have asked Doctors Bulson's permission to show a lantern slide of casts from the tracheobronchial tree in a case of tracheobronchial diphtheria. I saw the patient, a boy 18 years of age, in consultation with Dr. Clayton Giddings and Dr. John MacKay Mitchell of Philadelphia. One of these casts measured 8 inches in length and included membrane from

the subglottic larynx, trachea and bronchi. A cast was coughed up on three separate occasions, each cast after the administration of a large dose of diphtheria antitoxin. The boy was treated at home (being the son of a physician) and we were prepared to remove the cast bronchoscopically if the occasion arose. The patient expelled the cast spontaneously, and I may say that we carefully avoided the use of morphine or cough sedatives so that his reflexes would remain active. The boy made an uneventful recovery, with the development of no sequelae.

Dr. E. B. Swerdfeger, Denver: I wish to report the rather unusual case of a woman 65 years old, a grandmother, living in a home with a large number of grandchildren. She had follicular tonsillitis, and when we took a culture it was negative for diphtheria. But she did not get well as quickly as she should, and had difficulty in breathing, so we made a laryngoscopic examination and found membrane below the cord. I was successful in getting a culture there, and it proved positive. She was given large doses of antitoxin, and within a few hours she coughed up a complete cast of about six rings of the trachea. She got well without any untoward symptoms following that.

Dr. Millard F. Arbuckle, St. Louis: I would like to report a case of diphtheria of the bronchus which I saw a few weeks ago:

The patient was a 2-year-old boy. Seven days before admission to the hospital, after eating peanuts, he had a sudden attack of coughing and choking, which subsided in a few minutes, but was followed by mild wheezing respirations. The following day he became hoarse and began to have slight respiratory difficulty. Four days after the onset, there was constant wheezing and stridor accompanied by low grade fever. There were no attacks of cyanosis.

The family physician suspected foreign body and sent the patient to the hospital for bronchoscopy.

Past and family history were negative. Admission physical examination revealed an anxious two-year-old white boy with laryngeal stridor, barking cough and slight hoarseness. Temperature 38.6, pulse 104, respiration 45. The skin was clammy and subcyanotic. The pharynx was mildly injected. Breathing was a bit labored, inspiration being accompanied by intercostal retraction and exaggerated action of the neck muscles. In the upper lobe of the right lung breath sounds were very faint and there was wheezing, while the percussion note over this area was hyperresonant. Elsewhere the breath sounds were normal except for slight general obscuration by the laryngeal stridor. Remainder of the physical examination was not remarkable. Admission diagnoses were acute obstructive laryngitis and obstruction of the right upper bronchus.

The white count was 20,000, with 55% polymorphonuclear cells. Other laboratory findings were not remarkable.

Fluoroscopy of the chest revealed limited movement of the diaphragm on both sides, with emphysema of the right upper lobe suggesting partial obstruction. No foreign body was seen by fluoroscope or X-ray.

Direct laryngoscopy revealed no membrane. The false cords were slightly injected, the true cords normal in appearance and function. The subglottic area was inflamed and edematous.

Bronchoscopy revealed the larynx considerably swollen and somewhat difficult to enter. The mucous membrane everywhere was red and covered with pus in quantities sufficient to fill the bronchoscope. The mucous membrane of the right upper lobe bronchus was quite swollen so that the bronchus was almost occluded, and pus exuded from its narrowed opening. No foreign body was seen. Following bronchoscopy the breath sounds over the right upper lobe became decidedly louder and there was less wheezing.

Cultures of larynx and trachea were taken and returned in twelve hours as positive for diphtheria. Ten thousand units of diphtheria antitoxin were given intramuscularly and 4,000 units intravenously.

A tracheotomy was done several hours after bronchoscopy, with instant respiratory relief. The child then made an uneventful recovery.

DEAFNESS DUE TO PANCREATIC INSUFFICIENCY: SYMPTOMATOLOGY AND TREATMENT.*

MARK J. GOTTLIEB, M. D.,

NEW YORK.

The deafness under consideration is that which is produced by poisons circulating in the blood. These toxins originate from some focus distant from the ear.

In speaking of deafness of constitutional origin, or constitutional deafness, one must consider its various causes. Categorically, they may be presented as follows:

Poisons and drugs taken by mouth.

Infectious diseases, such as mumps, scarlet fever, diphtheria, typhoid fever, pneumonia, influenza, tuberculosis and syphilis.

Focal infections.

Disturbances of glands of internal secretion, such as dysfunction of the pituitary, thyroid, adrenal, ovary, testicle and parathyroid.

Circulatory diseases such as arteriosclerosis, cardiac and renal diseases.

Metabolic diseases such as gout and, particularly, diabetes.

Biliary dysfunction.

Pancreatic digestive dysfunction.

This treatise undertakes to present a study of lowering of the hearing capacity in cases wherein is found a disturbance of the secretion of the enzymes of the pancreas into the intestines. Associated with this is frequently found a biliary dysfunction. Disturbances of the biliary function can exist without impairment of the external secretion of the pancreas, but I am not sure that dissociation of the pancreatic digestive secretion can occur without the biliary disturbance. Gall bladder disease is frequently found in conjunction with this pancreatic disturbance, as will be seen during the progress of the presentation. That the insular portion of the pancreas is involved in the process is evident by the occasional finding of an increase in blood sugar above the

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high normal figure. Thus we find that reduction in hearing is found in conjunction with disturbances of the function of organs which are of vital importance to the bodily economy, namely, the pancreas and liver.

That deafness associated with the abnormal findings in the function of the pancreas and liver exists in no small majority of all forms of nonsuppurative deafness will be appreciated when the compilation of cases here presented is studied.

As the result of making audiometer readings on a large number of patients suffering with a variety of ailments, I find that many have a depreciation in acuity of hearing and sight. The loss in acuity of light perception was estimated by plotting the fields for form and color. Incidentally, I might say that Drury[1] was the first to call attention to the fact that contracted visual fields for form and color were encountered in cases of deafness of the nonsuppurative type. This observation was published in 1926. Gottlieb[2] made some independent observations on the same subject, which were published in 1928. The amount of reduction in hearing and seeing acuity was found to be dependent directly upon the character, severity and chronicity of the illness. The greatest and most constant losses in hearing are found in cases of diabetes. Next in line are diseases of the gall bladder and liver dysfunction. Equal to this is the hearing loss encountered in circulatory diseases. Cases of hyperthyroidism are found to have little loss except when complicated by myocarditis or nephritis, etc. Over-action of the thyroid gland is encountered quite often in association with biliary dysfunction. The patients examined did not complain of difficulty in hearing. In some cases the loss of hearing was great enough to warrant this symptom, but it seems that the disturbances produced by the primary illness precluded a complaint about hearing difficulties.

It has been my contention for some time that the cause of a large number of cases of hard of hearing is due to intestinal autointoxication. With this thought in mind, I[3] injected an extract of stool from certain cases of deafness into guinea pigs, using the stool of persons with normal hearing as controls. The preliminary report of this work has been published. In short, we obtained hemorrhagic and exudative lesions in the cochleæ of the guinea pigs so treated. Such lesions were not found in the controls. As a result of this work it was natural to investigate the pancreatic function in cases of deafness. As you know, the ex-

ternal secretion of the pancreas is the most important and powerful digestant that the body possesses. If the digestive capacity of this secretion is lowered below a certain point, the partially digested food will undergo bacterial decomposition and the products of this putrefaction may be absorbed into the blood stream and produce various forms and grades of poisoning. It is likely that the toxins affect all parts of the body, but we are more aware of the loss in hearing perception because it affects our efficiency, both economically and socially.

We have examined the pancreatic function of 38 cases of deafness. The methods used are as follows:

The blood for chemistry was taken after starvation for over 12 hours. The duodenal contents were removed after a period in which no food was taken. After the tube entered the duodenum, 30 cc. of 25 per cent magnesium sulphate was poured through the tube. After 15 minutes, duodenal drainage was allowed to proceed and a portion of the darkest bile stained fluid was submitted for examination.

Tryptic activity was determined by estimating the amount of 0.1 per cent casein solution that is digested by 1 cc. of duodenal fluid exposed for 15 minutes at a temperature of 38°. Gross' Method, Hawk, "Practical Physiological Chemistry," 8th edition, page 194.

Amylolytic activity was determined by the amount of digestion of 1 per cent starch solution by 1 cc. of duodenal contents for 30 minutes at 38°. Wohlgemuth: Biochemische Zeitschrift, 9, 1, 1908.

Steapsin.—The activity of steapsin is estimated by determining the amount in milligrams of fatty acids that are formed by hydrolysis, using olive oil emulsion, by 1 cc. of duodenal contents. The fatty acid is calculated in terms of oleic acid. J. Wohlgemuth: "Grundriss der Fermentmethoden," pages 100-103. (Kanitz.)

We are aware that the methods used do not produce consistent and accurate figures because of certain chemical and biologic factors. These have been very clearly set forth by McClure[4] et al., who has devised more accurate procedures for these estimations. Unfortunately, we in New York have not the facilities to perform the tests as set forth by McClure.

However, as will be shown later, we do not believe that the error in our figures is sufficiently great as to nullify the validity of the result of this investigation.

As the result of the examination of the duodenal contents of many patients, we have established low normal figures for good digestion. They are for trypsin, 10 cc; for amylopsin, 20 cc., and for steapsin, 50 mgms. We believe that these figures are too low, because patients with hearing loss who exhibit higher figures show improvement in hearing after taking pancreatic extract.

Of the 38 cases that show a reduction in hearing and whose pancreatic digestive activity has been estimated, we find that nine have digestive figures above the established low normal figures and 29 display figures that are below this standard.

Out of the total of 38 cases, 7 give evidence of having diseased gall bladder or biliary dysfunction. Four of these occur among the cases that have a pancreatic digestive figure higher than the low normal figure and three among those whose digestive activity is less than these figures. Four cases in this entire group have enlarged and over-active thyroids—these occur among the group that have a lowered pancreatic digestive capacity.

The greatest and most consistent reduction in pancreatic digestive activity occurs in the tryptic moiety, but severe losses of activity occur in the other fractions as well. For instance, there is no amylolytic digestion in one case, and only a trace in another. Likewise there is no digestion for fats in two cases. These have all been examined several times, and one of these (M. G.) was examined also by Dr. McClure, who confirmed our finding.

In seven cases the blood sugar values were found to be above the high normal figure—six of these occurred among the group wherein the low digestive figures were encountered. In only one case (A. V.) sugar was found in the urine.

It is evident that we have concerned ourselves with an investigation of the integrity of the acinous and insular portions of the pancreas in cases of deafness. The result has been that the majority of the cases examined have a paucity of pancreatic intestinal digestion in one or more elements, and high blood sugar figures are occasionally encountered combined with this dissociation.

All observers have been impressed with the fact that cases of nonsuppurative deafness occur in families, and there seems to be a hereditary predisposition to the affliction. Quite often, however, cases present no history of a familial tendency or hereditary trait. Nager (1928)[5] finds that hereditary or familial evidence

occurred in 58.6 per cent of his cases. There is a great variation in the figures relative to the incidence of other cases in the same family or the history of deafness in the antecedents. It is certain, however, that this occurrence is sufficiently common to claim our interest.

As was stated previously, the most severe and constant losses of hearing occur in cases of diabetes. By comparing the above findings with those in diabetes, it is evident that certain cases of deafness and diabetes apparently have elements in common. For instance, Dubnova and Izigson[6] claim that in diabetes the external as well as the internal secretory activity of the pancreas are impaired. Allen believes that the activities in the islands of Langerhans and the acinous tissue represent the result of an acute, subacute or chronic pancreatitis. Also that mild diabetes occurs when lesions of the pancreas are due to extension of the pathologic process from the biliary tract. McClure[4] and Jones claim that a dissociation of pancreatic digestive activity, similar to that which is shown here, is due to various grades of interstitial pancreatitis. Joslin shows that out of 2,900 cases of diabetes studied, 21 per cent had a familial history of the disease.

The incidence of deafness associated with pancreatic dysfunction is so great that it cannot be disregarded. Its association is not accidental. However, all cases of pancreatic disease do not go deaf. The explanation for this may be found in studying the difference in anatomy of various individuals. These differences are transmitted from parent to offspring. Polydactylitis occurs in families. Impacted wisdom teeth are more likely to occur in issues from parents whose molars were also impacted. The circulation of the cochlea varies in individuals. Nabeya[8] has shown that there are three types of circulation in the cochlea of man. In the first kind the arteria propria cochleæ is practically a terminal vessel from its source—there being only a few minute arborizations between it and the arteria vestibulocochleæ in the basal coil. A person with such a cochlear circulation is more liable to become deafened than one who has the second kind or the third kind of blood supply. There is no doubt that during toxemia of any origin the sensitiveness of nerves is diminished, and when an organ is especially vulnerable greater damage will result. It is possible that we inherit the type of circulation that our ancestors possessed, and this may explain the frequency of hereditary deafness. That hearing losses are frequently encoun-

tered in cases suffering with circulatory diseases is suggestive of a relation between auditory acuity and blood vessel integrity. This idea is further encouraged when on investigating the detailed symptoms of the cases in consideration we find that the tinnitus is made louder and more annoying, and that the hearing is less acute following coitus. Furthermore, following a short interval after awakening from sleep, the tinnitus is resumed with greater force than at any other time during the day, and this lasts for a short time. There is no doubt that profound changes occur in the circulation just prior to and following sexual intercourse and during the transition from sleeping to waking.

In the history of these cases we find that the hearing difficulty was preceded by an acute infectious disease, migraine headaches, gastrointestinal disturbances, or attacks of gall stone colic. Headache is a common complaint; it is usually bilateral and occurs often; at times it is accompanied by nausea and even vomiting. Occasionally patients will state that they have a feeling that a headache is constantly impending. Mild photophobia is a frequent symptom. It is painful for the patient to hear sudden raucous noises, particularly when applied to the ear most severely affected. The majority complain of slight spontaneous but inconstant pain which originates in the ear and travels along the anterior wall of the external auditory canal and radiates in diminishing intensity over the side of the face directly in front of the ear. Some patients complain of slight pain behind the ear. It is most likely to be present in the worst ear. Vertigo is not common, but when it enters into the picture it is distressing. Tinnitus is not always a symptom. It is, however, a frequent complaint. It is more intense and has a lower note in the ear most affected. At times the tinnitus is made up of a combination of tones and noises. The tinnitus is worse on reclining, but it is more intense directly after awakening. It is augmented following the sexual act. The pitch of the tinnitus becomes lower and the intensity greater as the hearing loss increases. When the hearing is improved the pitch becomes higher and softer. Loss of hearing is not sensed until hearing loss for 512, 1024 and 2048 D. V. sinks below 30 units. If the high and low notes are mainly depressed the loss is only occasionally appreciated. The loss in hearing is progressive and slow—years pass before appreciable damage is done. In some cases the patients become deaf within a short time after its apparent onset. Cases have been encoun-

tered wherein incapacitating deafness occurred within a period of a few weeks.

The appearance of the drum is the same as is seen in nonsuppurative deafness due to other causes. It is not characteristic. It may look normal, lusterless and retracted or may have areas of redness around the malleus and the periphery of the drum. The eustachian tubes are easily inflatable. The hearing may or may not be improved by inflation. At one sitting the hearing is better after inflation, and at another time the hearing is made worse. One ear may be improved by inflation and the other show a definite depreciation. By doing readings of the hearing before and after inflation in a large number of cases, we have concluded that inflation has no diagnostic significance. This also applies to nonsuppurative deafness in general. This does not, however, include cases of deafness due to adhesions and other local disease in the middle ear and the eustachian tube.

The character of the hearing curve does not follow a particular pattern. In some cases the drop is greater in the low notes than in the rest of the scale. The majority of patients, however, display a greater loss in the high notes than elsewhere. The bone conduction is usually shortened, but in some cases there is a prolongation.

The depreciation in hearing does not progress in a straight line. It drops and then improves—each drop is lower than the previous depression. If improvement occurs, it follows the same scheme but in reverse order.

Contracted visual fields for form and color, and color alone, are commonly found in this condition. Not all cases show such contraction. Some have such severe contraction that one may expect the patient to complain of funnel vision. No such symptom has been noted, however.

The Wassermann reaction was found to be negative in all of the cases included in the chart. We do find abnormalities in the blood chemistry figures in these cases. The uric acid is usually higher than the high normal, but the figure is rarely above 5 mgms. The calcium figures are usually abnormal. It may be below or above the normal standard. The significance of these findings is not as yet understood.

Usually patients suffering with deafness and pancreatic dysfunction are undernourished and underweight. In a few of the

cases studied the weight is normal for the height and age of the patient.

The treatment of deafness due to pancreatic dysfunction is essentially an effort to institute proper pancreatic digestion where absence or deficient digestion exist. When liver dysfunction or gall bladder disease is also present, treatment to correct this may also be instituted.

Essentially the treatment consists in an effort to introduce into the duodenum sufficiently active pancreatic extract and in such quantities that it will be sufficient to digest the food completely in the intestine. As the result of a considerable effort, I have caused to be produced a glycerinated extract—suspension of whole fresh pancreas. The product is standardized and its digestive capacity is never less than 200 cc. for trypsin (method of Gross), 500 cc. for amylopsin (method of Wohlgemuth) and 2,500 mgms. for steapsin (method of Kanitz). The ideal method of introducing the product into the small intestine is by means of the duodenal tube. Three teaspoonfuls of the extract are diluted with four ounces of water and the mixture is alkalinized with twenty grains of bicarbonate of soda. It is introduced directly into the duodenum at body temperature and should be repeated three times weekly. This method of treatment is particularly applicable to cases that have an absence of pancreatic digestion in one or more elements. There is no doubt that pancreatic extract when given by mouth into the stomach suffers a depreciation through the action of the gastric juice, and, in cases of absolute deficiency, to obtain results the extract must reach the intestine unimpaired.

In cases where there is a lowering of the pancreatic activity in one or more of the enzymes and not a complete loss, pancreatic extract when given by mouth works very well. A certain percentage of the pancreatic extract survives the gastric digestion. The method of administration is two teaspoonfuls of pancreatic extract in one-half glass of water, three times daily, 20 minutes or more before eating. This mixture begins to appear in the duodenum within 10 to 15 minutes after administration when taken on an empty stomach. No food should be taken between meals or before bedtime.

The diet should be regulated in accordance with the findings. When the trypsin portion is absent or lowered the amount of

proteins should be limited. When the other enzymes are deficient the foods that are digested by each should also be curtailed.

In cases where the gall bladder or liver is also found to be diseased, it is advisable that a duodenal drainage be done once or twice weekly, using 25 per cent magnesium sulphate as a hepatic stimulant, and when this is finished the pancreatic extract mixture mentioned above should be instilled into the duodenum and before each meal the pancreatic extract should be given by mouth.

This treatment should be continuous, because as soon as it is interrupted any gain in hearing which resulted from the treatment is quickly lost.

For some reason not understood, pancreatic extract is constipating, and to offset this it is advisable for the patient to take an enema every other day and a mild cathartic once weekly. Strong catharcis is interdicted because it washes from the intestine valuable digestive juices. Such catharcis is always followed by a definite hearing loss.

Alcohol, tobacco and excessive fatigue have a deleterious effect on the hearing of those who are deafened. This should be avoided.

In all, 23 cases have been treated, 19 of which show definite improvement in hearing.

In some of the cases the gain in hearing acuity was surprisingly great. The four cases that did not show improvement are those whose digestive figures were either nothing for one or more of the enzymes, or the digestion was so slight as to be negligible. Treatment is now being instituted in these cases by means of instilling the pancreatic extract directly into the duodenum.

REPORT OF CASES.

Cases No. 1 to No. 8, inclusive, have not been treated with pancreatic extract, because it was felt that the digestive figures were sufficiently high to warrant the assumption that there was not a deficiency. However, case No. 6 was treated experimentally with pancreatic extract for five months to determine whether such treatment would influence the hearing of a case with good digestive figures. The hearing was not improved.

Cases Nos. 13, 16, 20, 25, 26, 32 and 33 have either received no treatment or the treatment has been too recent to obtain a result.

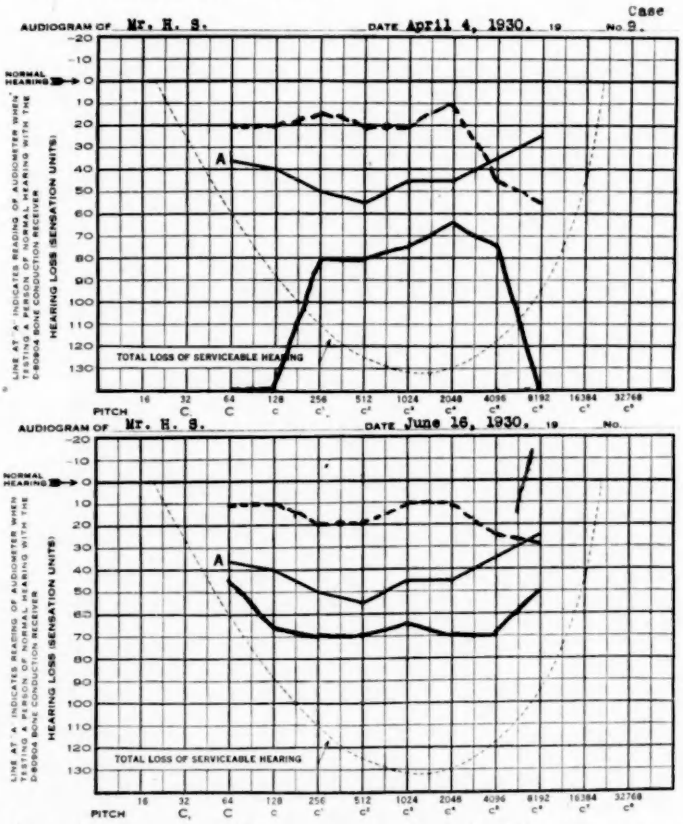
Cases Nos. 17, 18, 37 and 38 did not respond to treatment and are to be considered failures. All of these cases have such low digestive figures in one or more of the pancreatic enzymes that it is felt that nothing short of administration of the medicament directly into the duodenum will be of any service to them.

Cases Nos. 19, 24, 34 and 35 were reported in the preliminary report of February, 1929.[9] All of these patients have shown a consistent improvement in hearing. The reason why they have not been reported graphically is because during their treatment the author changed from the old method of testing hearing to the audiometer, and therefore satisfactory comparisons could not be made.

The remaining cases are herein given with necessary details.

In the illustrative audiograms the solid line indicates the right ear, and the broken line the left.

Case No. 9. Mr. H. S. September 20, 1929; 40 years. Has had difficulty in hearing since three months, particularly in the right ear. Has had cramps in abdomen for five years. Constantly constipated; occasional headaches. No history of deafness in family. Right drum is apparently normal, left drum thin in places and retracted. Pus can be expressed from both tonsils. Has thickened and deviated septum. Eye fields are normal. Hearing is not changed by inflation. Rinné is negative on both sides, bone conduction is reduced on both sides. The laboratory reports the following: Urine, negative; Wassermann, negative; sugar in blood, 82 mgms.; uric acid, 3.2 mgms.; calcium, 10.4 mgms. The digestive capacity of the duodenal contents shows trypsin, 17 cc.; amylopsin, 33 cc.; steapsin, 634 mgms. The basal metabolism is —2%. X-ray of gall bladder shows evidence of pericholecystitic adhesions.


AUDIOGRAM OF Mr. H. S. DATE April 4, 1930. 19 Case No. 9.
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
A
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
AUDIOGRAM OF Mr. H. S. DATE June 16, 1930. 19 No.
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
A
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

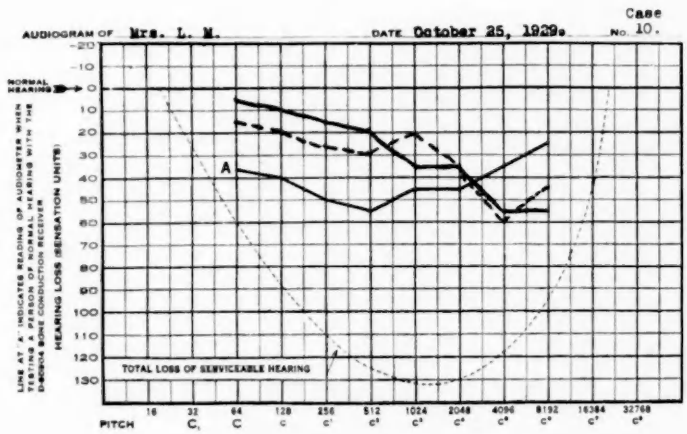
The patient began to take treatment on April 4, 1930. The reason for the delay in treatment was because it was felt that his digestive fluid had sufficient power to warrant the assumption that the cause was not to be found in the pancreas. The result of the treatment from April 4th to June 16th is graphically demonstrated in the following.

From the time of admission in September, 1929, to the day when treatment was begun, his hearing, especially on the right side, gradually became

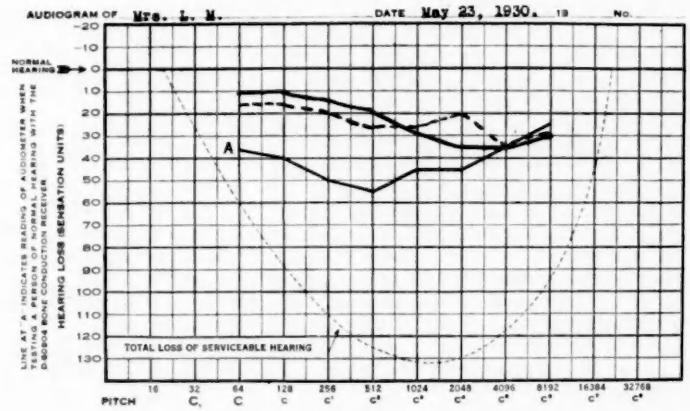
worse. He now says that since taking pancreatic extract, his digestion is much improved, but he is not quite sure that there is an improvement in hearing.

Case No. 10. Mrs. L. M. February 26, 1929; 26 years. Has had difficulty in hearing for about 1½ years. Believes that the onset occurred much earlier, as this came on gradually. In early childhood had scarlet fever followed by nephritis. Has always been thin. Mother is deaf. (See report of Mrs. K. K., Case No. 35.) Drums exceedingly transparent. Hearing not improved by inflation. Visual fields normal. The laboratory reports the following: Urine, negative; Wassermann, negative; sugar in blood, 101.3 mgms.; calcium, 13.6 mgms. The digestive activity of the duodenal fluid shows trypsin, 10 cc.; amylopsin, 25 cc.; steapsin, 464 mgms.

Since taking pancreatic extract, this patient has put on weight. She claims a general improvement in health and in hearing.

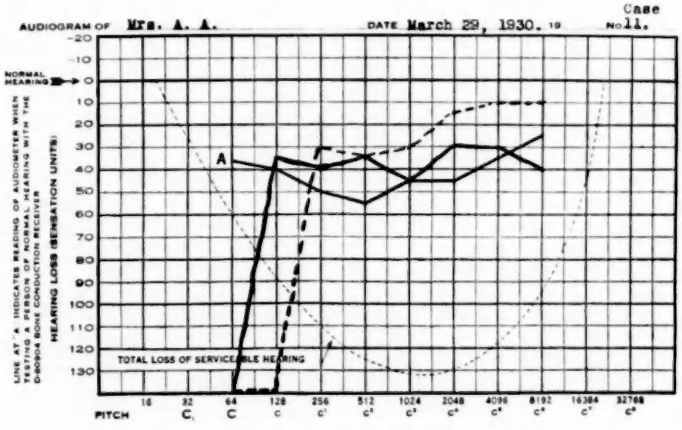

Case
AUDIOGRAM OF Mrs. L. M. DATE October 25, 1929. No. 10.
NORMAL HEARING
LINE AT 'A' INDICATES READING OF AUDIOMETER WHEN TESTING A PERSON OF NORMAL HEARING WITH THE DARROW BONE CONDUCTION RECEIVER
HEARING LOSS (SENSATION UNITS)
A
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
C1 C c c1 c2 c3 c4 c5 c6 c7 c8

Pancreatic extract and diet prescribed.

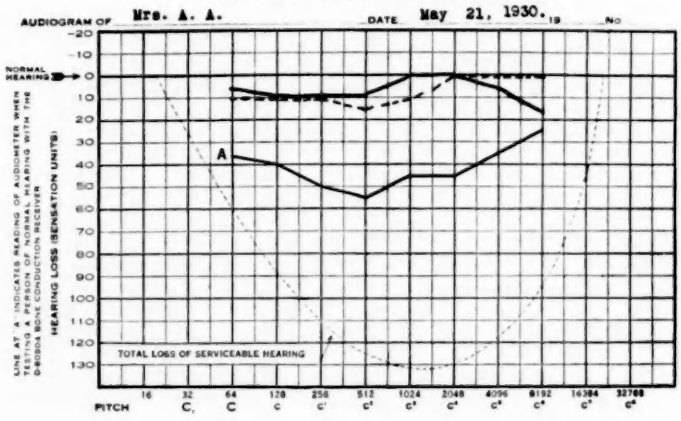

AUDIOGRAM OF Mrs. L. M. DATE May 23, 1930. No.
NORMAL HEARING
LINE AT A INDICATES READING OF AUDIOMETER WHEN TESTING A PERSON OF NORMAL HEARING WITH THE DARROW BONE CONDUCTION RECEIVER
HEARING LOSS (SENSATION UNITS)
A
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
C1 C c c1 c2 c3 c4 c5 c6 c7 c8

Case No. 11. Mrs. A. A. March 29, 1930; 38 years. For the past four years has noticed that she has difficulty in hearing, especially the low notes. No family history of deafness. For the past one and one-half years has had diarrhea accompanied by occasional attacks of pain and nausea. On several occasions saw blood in her stool. Has lost considerable weight. During childhood had some intestinal trouble which lasted a long time. The patient is exceedingly thin, and has prolapse of both kidneys. The laboratory reports the following: Urine, negative; Wassermann, negative; sugar in blood, 111 mgms.; uric acid, 1.2 mgms.; calcium, 15 mgms. The digestive activity of the duodenal fluid shows trypsin, 10 cc.; amylopsin, 50 cc.; steapsin, 182 mgms.

Since receiving treatment this patient has put on weight, and claims that all of her digestive symptoms have disappeared and that she has no difficulty in hearing.

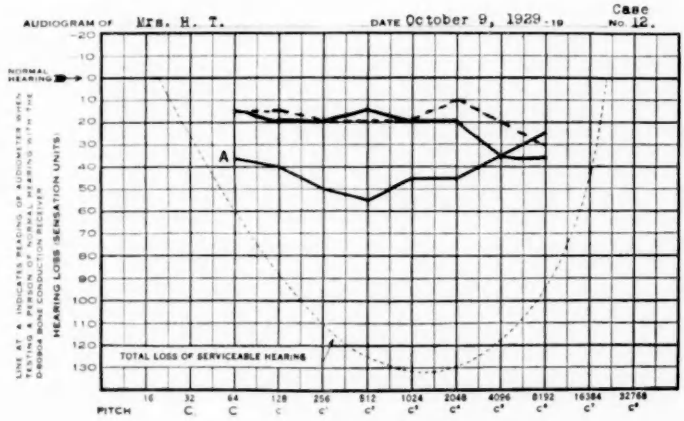


Diet and pancreatic extract prescribed.

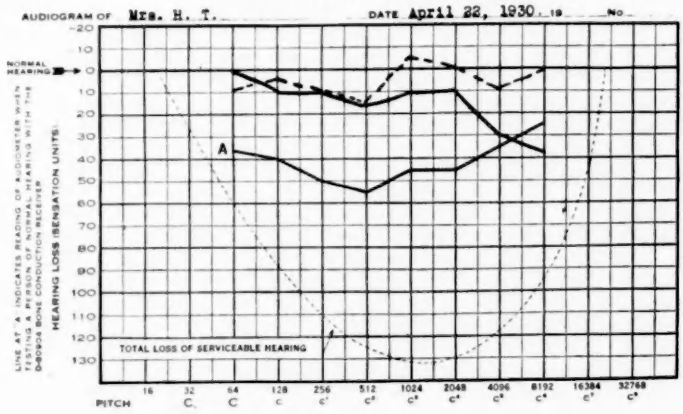


Case No. 12. Mrs. H. T. October 8, 1929; 54 years. For past five years has had noises in the right ear, which becomes worse on reclining. At times this is high pitched and at other times low. Pneumonia and typhoid fever at the age of 9 years. Nine years ago had upset of the stomach. No history of heredity. Hearing improved upon inflation. The laboratory reports the following: Urine, negative; Wassermann, negative; sugar in blood, 116 mgms.; uric acid, 1.3 mgms.; calcium, 9.6 mgms. The digestive activity of the duodenal fluid shows trypsin, 14 cc.; amylopsin, 10 cc.; steapsin, 155 mgms.

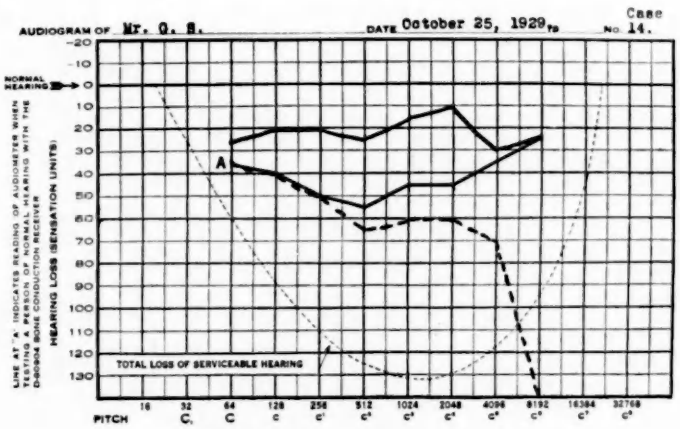
The patient claims that the tinnitus has disappeared since she has been under treatment.



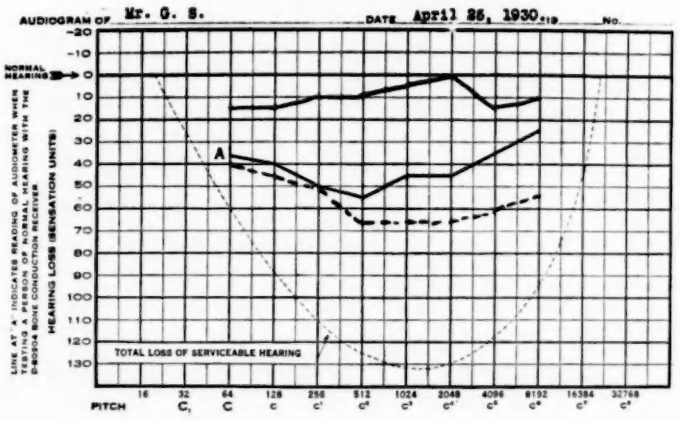
Diet and pancreatic extract prescribed.



Case No. 14. Mr. G. S. October 18, 1929; 46 years. This patient does not know when this condition began, but for past three months has had marked increase and difficulty in hearing and also dizziness. At times the dizziness is so severe as to cause him to fall. Has feeling of fullness for a long while after eating. At times has attacks of diarrhea. One brother is deaf. Has diseased tonsils. Visual fields slightly contracted for color. The laboratory reports the following: Urine, negative; Wassermann, negative; sugar in blood, 86 mgms.; uric acid, 4.5 mgms.; calcium, 7.6 mgms. Basal metabolism, +17%; kidney function normal. The digestive activity of the duodenal fluid shows trypsin, 14 cc.; amylopsin, 7 cc.; steapsin, 704 mgms. X-ray of gall bladder, normal.



Pancreatic extract and diet prescribed.

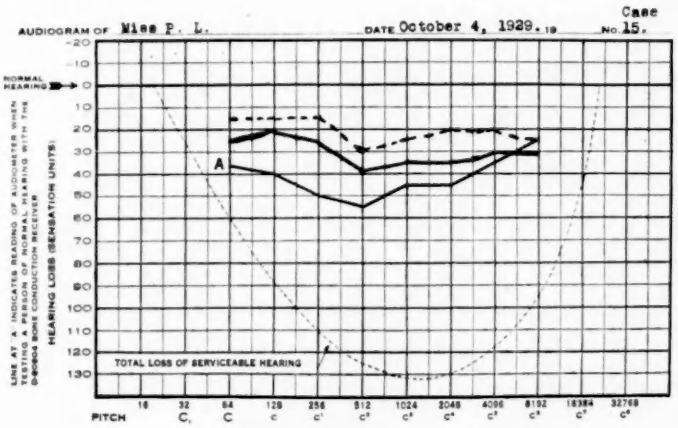


Case No. 15. Miss P. L. October 4, 1929; 24 years. For the past three or four years the patient noticed her inability to hear well; this has been progressive. For the past month has had repeated headaches. Has a feeling of fullness for a long time after eating. No history of heredity. For past two years has had watery nasal discharge; has had several nasal operations. The drums are retracted and lusterless. Small pieces of tonsils can be seen on both sides. Hearing improved upon inflation. Visual fields slightly contracted for color, especially on the left side. The laboratory reports the following: Urine, negative; Wassermann, negative; sugar in blood, 82 mgms.; uric acid, 3.2 mgms.; calcium, 9.4 mgms. The digestive capacity of the duodenal fluid showed trypsin, 12 cc.; amylopsin, 5 cc.; steapsin, 732 mgms.

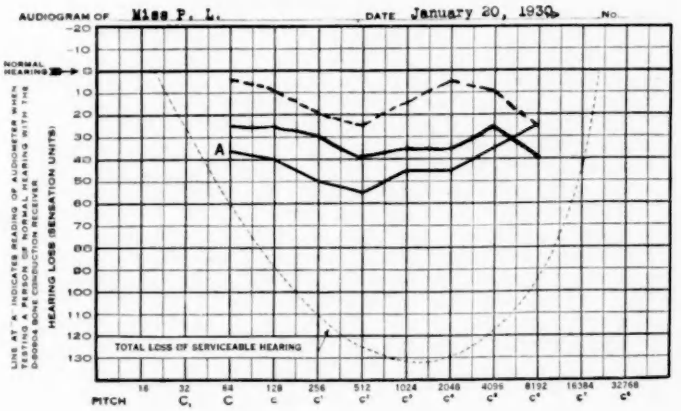
Rinné on the right side, negative.

Rinné on the left side, positive.

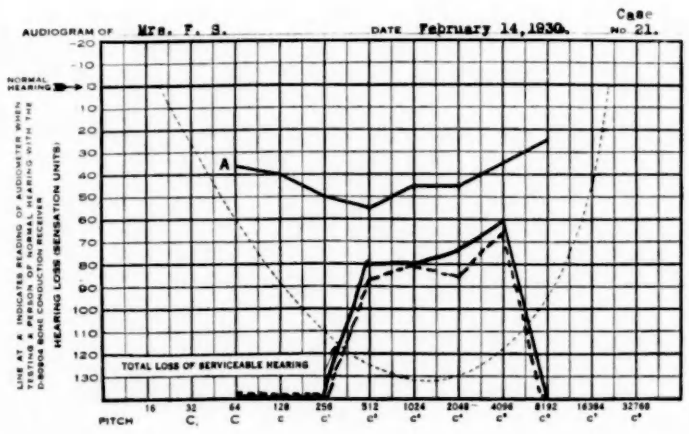
Bone conduction, shortened.

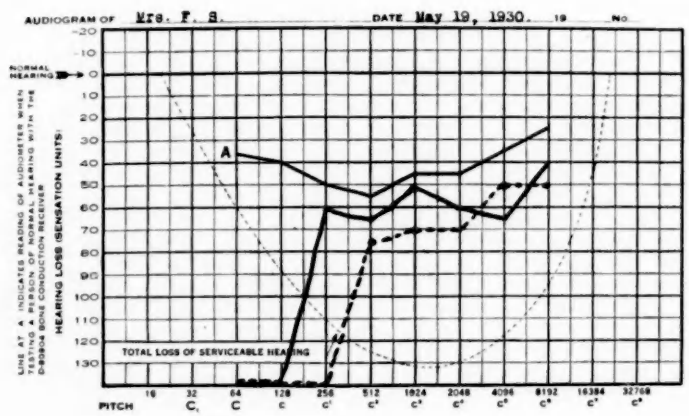


Diet and pancreatic extract prescribed.

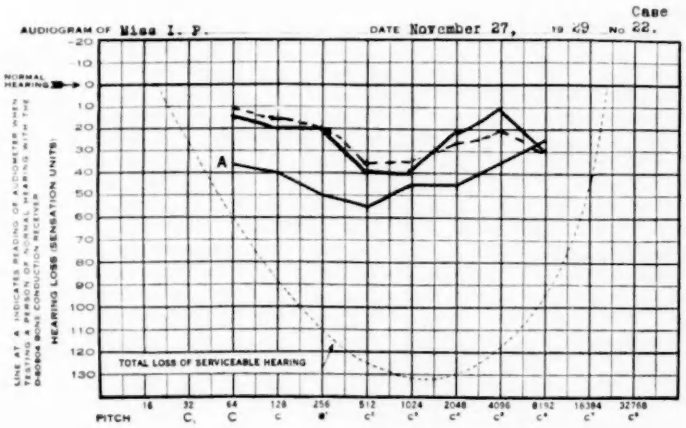


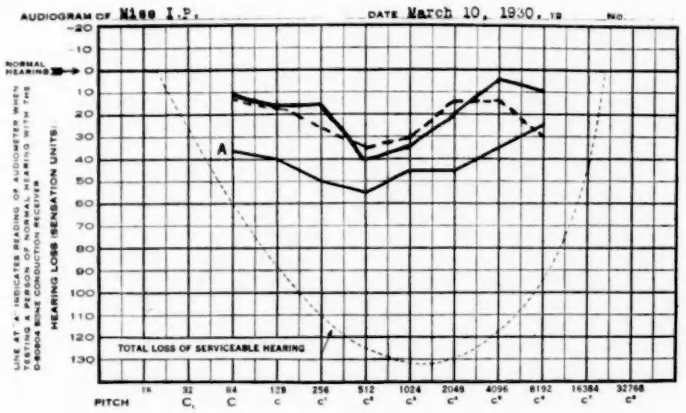
Case No. 21. Mrs. F. S. February 17, 1930; 38 years. Patient noticed progressive loss of hearing with both ears for past eight years. During that period she had been troubled with her stomach, having attacks of vomiting and constipation accompanied by headaches. Has had tinnitus in both ears for eight years; this stopped three years ago and now reappeared. Has occasional attacks of dizziness. Patient has slight enlargement of thyroid and tremor. Has high deviation of septum; drums lusterless. Hearing slightly improved on inflation. Rinné, negative on both sides; bone conduction shortened on both sides. Visual fields, normal. The laboratory reports the following: Urine, negative; Wassermann, negative; sugar in blood, 82 mgms. The digestive capacity of the duodenal fluid shows trypsin, 5 cc.; amylopsin, 107 cc., and steapsin, 396 mgms. Basal metabolism on February 18th was +18% and on February 26th +1%. X-ray of gall bladder was normal.





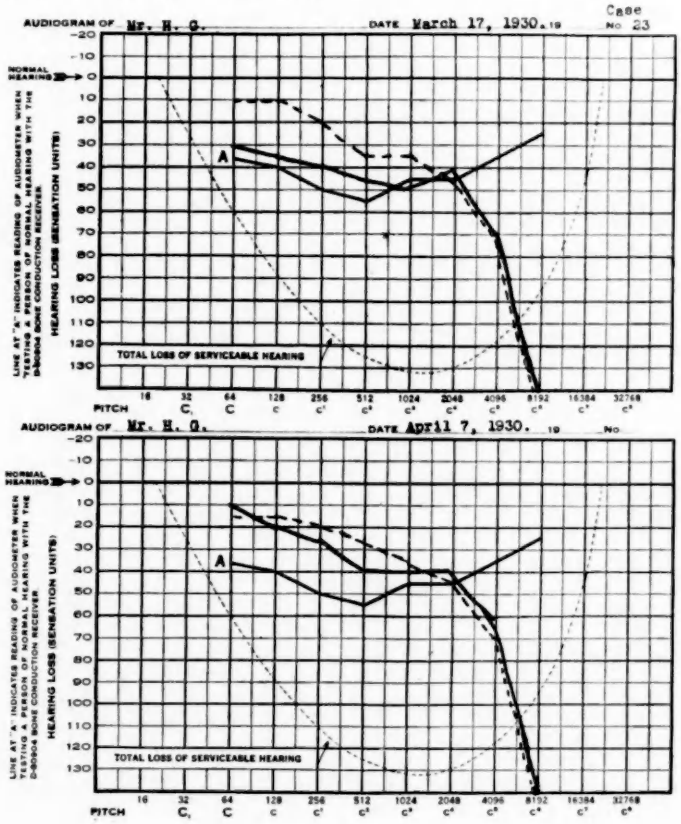
Case No. 22. Miss I. P. November 15, 1929; 30 years. Has had difficulty in hearing for past ten years, becoming progressively worse. At the age of ten had diphtheria and in 1918 had influenza, which has recurred several times since. Has headaches previous to menstruation. At times has headaches that last for weeks. Has attacks of dizziness. One sister is deaf. (See report of M. P., Case No. 4.) Hearing improved by inflation. Form and color fields are moderately contracted. The laboratory reports the following: Urine, negative; Wassermann, negative; sugar in blood, 103.3 mgms.; uric acid, 2.8 mgms.; calcium, 13 mgms. The digestive capacity of the duodenal fluid shows trypsin, 5 cc.; amylopsin, 80 cc., and steapsin, 185.2 mgms.





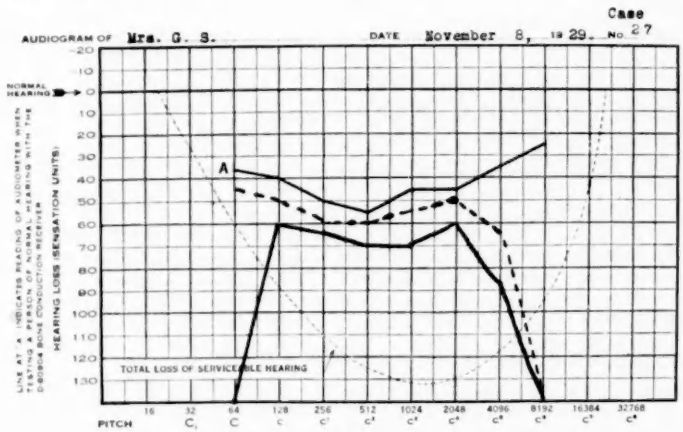
Case No. 23. Mr. H. G. September 7, 1929; 20 years. Patient noticed that he did not hear very well six months previous to this consultation. Occasionally he has heartburn. No history of heredity. Has chronic pharyngitis. Pink blush can be seen through each drum. At times improvement can be seen upon inflation, and at other sittings it is made worse. The hearing test for conversation for the right side was 6 feet and on the left, 10 feet. The watch could be heard at concha on both sides. Rinné was positive on both sides. He was treated by inflation between September 9, 1929, and November 18, 1929. At times his hearing, particularly on the left side, was markedly improved by inflation. On February 1, 1930, when he was put under treatment with pancreatic extract, his hearing for conversation was 5½ feet on the right and 11 feet on the left ear. Since then he has consulted this office only on March 17 and April 7, 1930, and the two audiograms of these dates follow. The time that this patient has been under treatment is entirely too short for the results to be of any value.

The laboratory reports the following: Urine, negative; Wassermann, negative; sugar in blood, 118.6 mgms.; uric acid 1.9 mgms.; calcium, 16 mgms. The digestive capacity of the duodenal fluid shows trypsin, 4 cc.; amylopsin, 180 cc., and steapsin, 650.

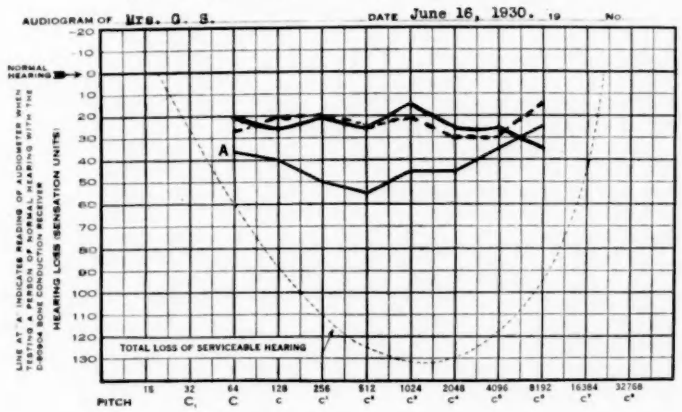

AUDIOGRAM OF Mr. H. G.
DATE March 17, 1930.
Case No. 23
NORMAL HEARING
LINE AT "A" INDICATES READING OF AUDIOMETER WHEN TESTING A PERSON OF NORMAL HEARING WITH THE D-80904 BONE CONDUCTION RECEIVER.
HEARING LOSS (SENSATION UNITS)
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
AUDIOGRAM OF Mr. H. G.
DATE April 7, 1930.
NO.
NORMAL HEARING
LINE AT "A" INDICATES READING OF AUDIOMETER WHEN TESTING A PERSON OF NORMAL HEARING WITH THE D-80904 BONE CONDUCTION RECEIVER.
HEARING LOSS (SENSATION UNITS)
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

Case No. 27. Mrs. G. S. 50 years. November 8, 1929. For the past year this patient has noticed increasing deafness in both ears combined with tinnitus and vertigo. Has had attacks of vomiting following vertigo. Ringing in the ear first occurs, then dizziness and from two to six hours later, vomiting. Has flashes before eyes, headaches seldom occur. Has deviated septum to right, polyps in the region of the left middle turbinate, postnasal discharge of mucopurulent material. Tonsils diseased; drums lusterless. No history of deafness in family. Hearing slightly improved on inflation. Has extreme concentric contraction of the form and color fields. Has incarcerated iris in left eye. The laboratory reports the following: Urine, normal; Wassermann, negative; sugar in blood, 96 mgms. per 100 cc.; basal metabolism, +6%. The digestive capacity of the duodenal fluid is trypsin, 2.5 cc.; amylopsin, 125 cc., and steapsin 591 mgms.

The following is a graphic record of the progress of this case. In addition to the improvement in hearing, a definite improvement is shown also in the visual fields. Patient still complains of noises in both ears.


Case
AUDIOGRAM OF Mrs. G. S.
DATE November 8, 1929 No. 27
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
A
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
16 32 64 128 256 512 1024 2048 4096 8192 16384 32768
C1 C c c1 c2 c3 c4 c5 c6 c7 c8

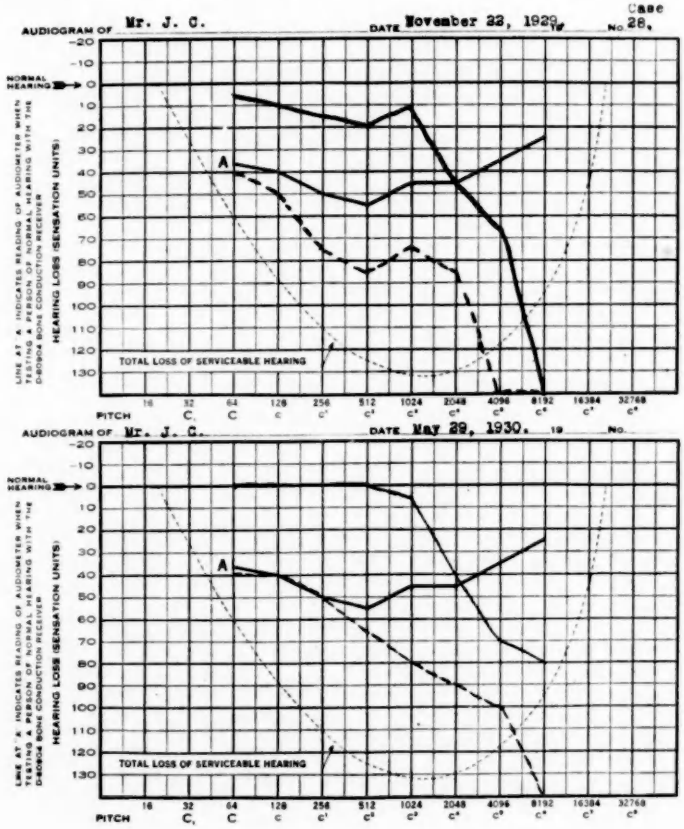
Diet and pancreatic extract prescribed- November 30, 1929.


AUDIOGRAM OF Mrs. G. S.
DATE June 16, 1930. 19 No.
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
A
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
16 32 64 128 256 512 1024 2048 4096 8192 16384 32768
C1 C c c1 c2 c3 c4 c5 c6 c7 c8

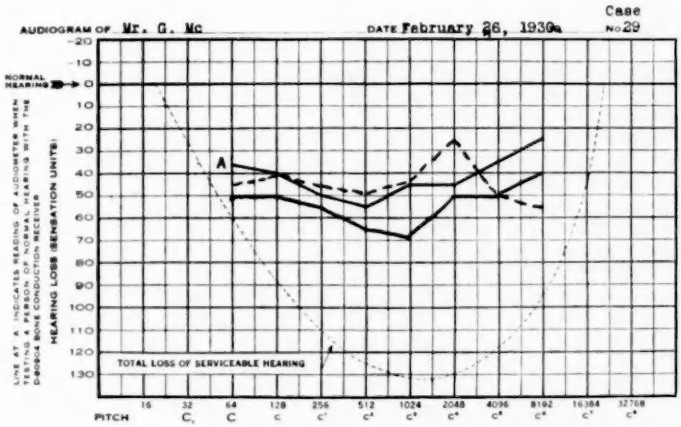
Case No. 28. Mr. J. C. November 19, 1929; 53 years. (Patient of Dr. Samuel J. Kopetzky.) The patient's chief complaint, distortion of pitch and clarity of sound in left ear, for the past ten years. For the past three years this has been much worse. Has had several attacks of severe sound distortion since then.

During January, 1930, following an attack of influenza, this sound distortion has been continuous. Has had trouble with his intestines and gall bladder since the past six years; has had periods of continuous diarrhoea. His father, paternal uncle and one brother are deaf. Patient is slightly improved on inflation. The bone conduction is shortened in both ears. Rinné is positive on the right side and negative on the left. The visual fields are slightly contracted for color. There is blurring of the margin of the left optic disc, the arteries are thin and the veins are a bit fuller than normal. The laboratory reports the following: Urine, negative; Wassermann, negative; sugar in blood, 81. mgms.; uric acid, 2.3 mgms.; calcium 9.8 mgms. Basal metabolism, —6%. The digestive capacity of the duodenal fluid is trypsin, 2.5 cc.; amylopsin, 30 cc., and steapsin, 394 mgms. The X-ray of the gall bladder with dye shows normal filling and emptying.

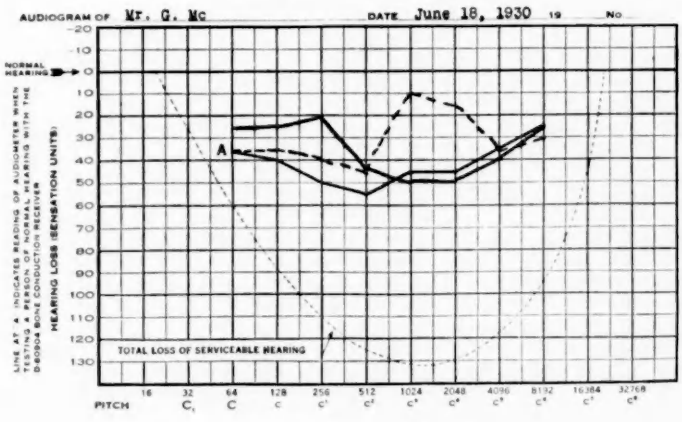
This patient has been taking treatment of duodenal drainage and administration of pancreatic extract by duodenal tube intermittently since his discharge from the hospital on the 23rd of November, 1929.



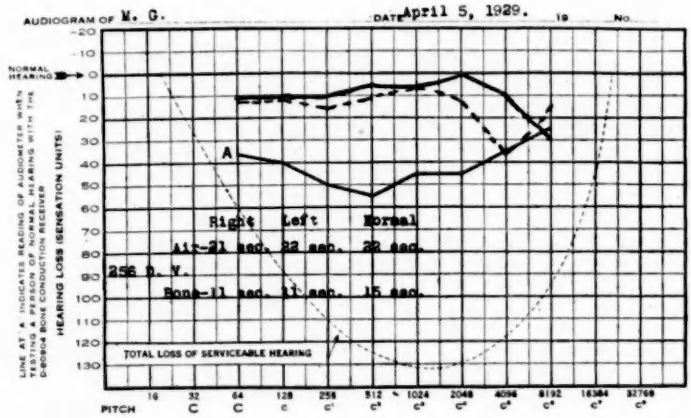
Case No. 29. Mr. G. Mc. February 26, 1930; 48 years. Has had deafness, both ears, since the age of 10, the hearing remaining stationary for thirty years, then became worse. Recently has had ringing in ears. At the age of 13 had diphtheria. Has been under treatment for duodenal ulcers for the past eight years. During the last year has had occasional discomfort four to five hours after eating. For a short time previous to this consultation has had a discharge from the left ear. Mother deaf at the age of 50. Brother stone deaf for forty years. Right drum has normal appearance, left drum retracted, and from a small perforation in the posterior inferior quadrant serosanguinous fluid is seen to exude. Tonsillar stumps in both fossae. The hearing of the right ear was improved upon inflation, and the left ear made worse. The laboratory reports the following: Urine, negative; Wassermann, negative; sugar in blood, 103.3 mgms.; uric acid, 2.8 mgms.; calcium, 17 mgms. The digestive activity of the duodenal fluid shows trypsin, 2.5 cc.; amylopsin, 83.3 cc.; steapsin, 108 cc.


AUDIOGRAM OF Mr. G. Mc
DATE February 26, 1930
Case No. 29
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
A
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
16 32 64 128 256 512 1024 2048 4096 8192 16384 32768
C1 C c c1 c2 c3 c4 c5 c6 c7 c8

Diet and pancreatic extract prescribed.


AUDIOGRAM OF Mr. G. Mc
DATE June 18, 1930
NO.
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
A
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
16 32 64 128 256 512 1024 2048 4096 8192 16384 32768
C1 C c c1 c2 c3 c4 c5 c6 c7 c8

Case No. 30. M. G.; 43 years. For about one year prior to April, 1929, the patient noticed that he had a sensation of fullness for a long time after eating, and that he was becoming habitually constipated. In November, 1929, he suddenly developed a tinnitus of the right ear which would disappear during the day and reappear on reclining. At times there were faint suggestions of tinnitus in the left ear. On April 5, 1929, his hearing was examined by Dr. Ralph Almour, giving the following record:

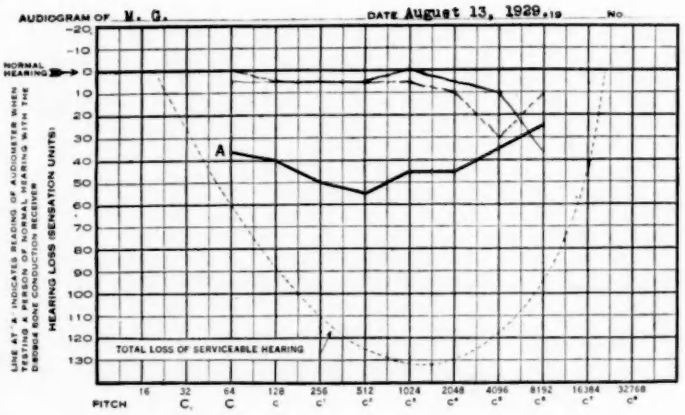


May 21, 1929, the duodenal contents were examined and showed: Trypsin, a trace; amylopsin, 75 cc., and steapsin, 0.

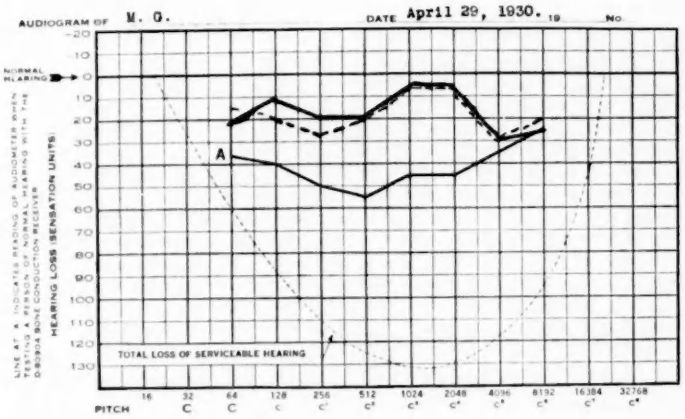
May 24, 1929, the duodenal contents gave the following result: Trypsin, 2.5 cc.; amylopsin, 50 cc., and steapsin, a faint trace.

The laboratory report is as follows: Urine, negative; Wassermann, negative; sugar in blood, 82.4 mgms.; uric acid, 1.6 mgms; calcium, 7.4 mgms.

Treatment was started by taking pancreatic extract by duodenal tube, three times weekly, and a limitation of proteins and fats in the diet. This was continued until August 12, 1929, when the following audiogram was obtained:



From that time until April *22*, 1930, pancreatic extract was taken by mouth with the result that there was a definite depreciation of hearing as shown in the following record:



No pancreatic extract was taken between April *22* and April *29*. On April 28th this patient consulted Dr. Charles W. McClure of Boston, who obtained a specimen of his duodenal contents with the following result: Trypsin, 2.5 cc.; amylopsin, 4.2 cc.; steapsin, 0.

Dr. McClure performs these tests in a different manner than that which is indicated in the body of this paper. His norms are: Trypsin, *2* mgms. up; amylopsin, 1 mgms. up; steapsin, 1 cc. up.

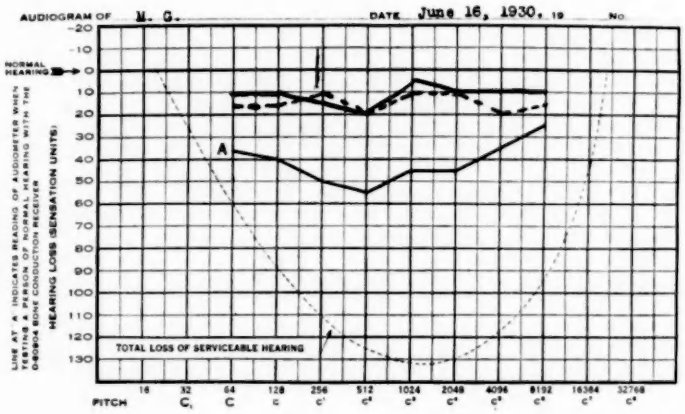
While the tube was in situ and after a specimen had been obtained for examination, two teaspoonfuls of pancreatic extract was taken into the stomach. This was done for the purpose of determining how much unimpaired pancreatic extract reaches the intestine when taken by mouth. The collection was obtained at two intervals of one-half hour each after the emulsion was administered. The result of the first half-hour period is as follows: Trypsin, 1.8 mgms.; amylopsin, 1.6 mgms.; steapsin, 1.1 cc. The result of the second half-hour period is as follows Trypsin, 2.5 mgms.; amylopsin, 3.5 mgms.; steapsin, 0.2 cc.

This indicates that some of the pancreatic extract when taken by mouth does arrive in the duodenum in an active form, but it appears that in this case the amount necessary to replace the deficiency was not enough when taken by mouth.

Dr. McClure also reported on the examination of other items in the duodenal fluid which are of importance in determining the function of the biliary system. The result is here given:

Furfurol no. 28 (normal no. 80-100).
Cholestrol 29.5 (normal 30 mgms.).
Alcohol insoluble pigment 0.7 (normal 7 mgms.).
Alcohol soluble pigment 5.0 (normal 8 mgms.).

Dr. McClure's opinion is that this patient is suffering with a pancreatic insufficiency which is related to a demonstrated hepatic dysfunction. Since this examination this patient has been taking pancreatic extract by mouth, and is also having a weekly gall bladder drainage, and twice weekly pancreatic extract is instilled directly into the duodenum with the result that the following audiogram is presented, showing an improvement over the hearing of April 29th, 1930:

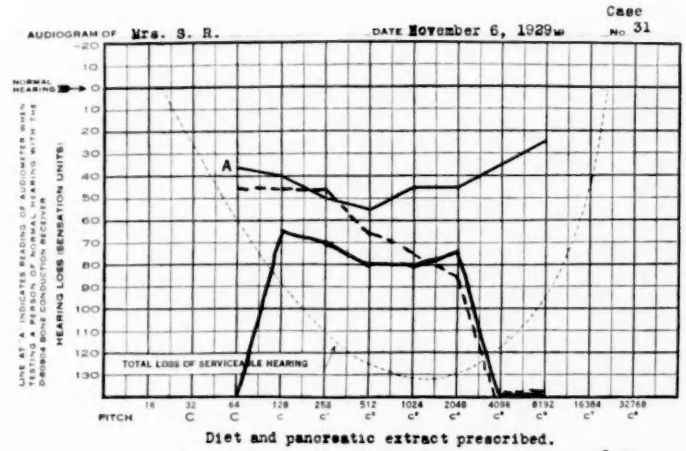


The visual fields were examined on May 7, 1930, and showed a slight contraction for color, especially on the right side.

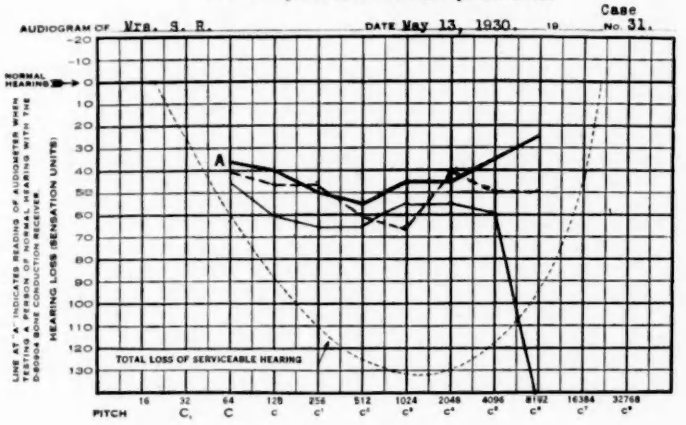
This represents an early case and suggests that the digestive insuffciency precedes the beginning of the hearing loss.

Case No. 31. Mrs. S. R. November 6, 1929; 39 years. This patient had influenza nine years ago. Since then has had a progressive loss in hearing. Has tinnitus in right ear. Two years ago was operated upon for gall stones. At present bowels regular, appetite good. Hearing improved slightly upon inflation. No history of deafness in family. The examination of the nose, throat and ears shows an apparently normal condition. The form and color fields show no definite abnormality. The laboratory reports the following: Urine, negative; sugar in blood, 90 mgms. The digestive capacity of the duodenal fluid showed trypsin, 2.5 cc.; amylopsin, 61 cc.; steapsin, 286 mgms. Rinné negative on both sides. Bone conduction, shortened.

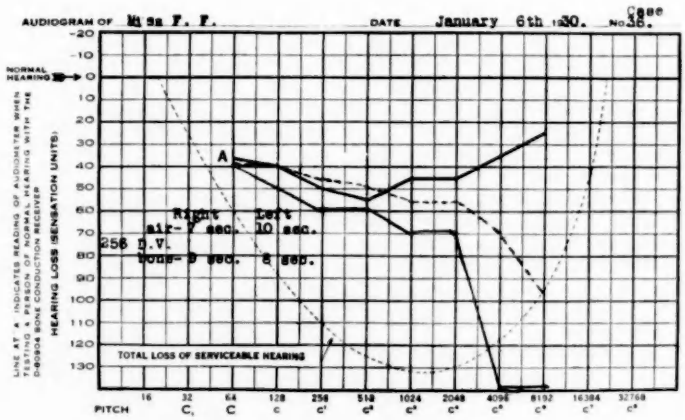
This patient has not taken her treatment regularly.


AUDIOGRAM OF Mrs. S. R.
DATE November 6, 1929
Case No. 31
NORMAL HEARING
LINE AT 'A' INDICATES READING OF AUDIOMETER WHEN TESTING A PERSON OF NORMAL HEARING WITH THE DeSOTO BONE CONDUCTION RECEIVER
HEARING LOSS (SENSATION UNITS)
A
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
C C c c' c² c³ c⁴ c⁵ c⁶ c⁷ c⁸

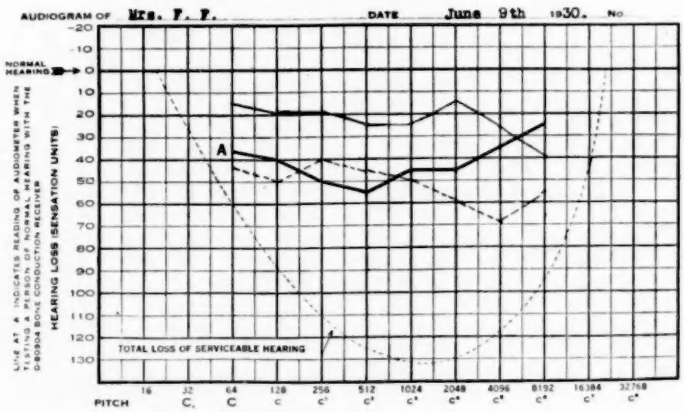
Diet and pancreatic extract prescribed.


AUDIOGRAM OF Mrs. S. R.
DATE May 13, 1930.
Case No. 31.
NORMAL HEARING
LINE AT 'A' INDICATES READING OF AUDIOMETER WHEN TESTING A PERSON OF NORMAL HEARING WITH THE DeSOTO BONE CONDUCTION RECEIVER
HEARING LOSS (SENSATION UNITS)
A
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
C₁ C c c' c² c³ c⁴ c⁵ c⁶ c⁷ c⁸

Case No. 36. Miss F. F. January 6, 1930; 25 years. For the past four years has had difficulty in hearing, accompanied by tinnitus. Has severe frontal headaches. Has had nasal discharge for past four years. Has had occasional food regurgitation. Slight contraction of form and color fields were found. Hearing not improved upon inflation. The laboratory reports the following: Urine, negative; Wassermann, negative; sugar in blood, 83 mgms. per 100 cc.; basal metabolism, 12%. The digestive capacity of the duodenal fluid is trypsin, 1 cc.; amylopsin, 62½ cc., and steapsin, 220 mgms. The X-ray of the gall bladder with dye suggests a pathological gall bladder.


AUDIOGRAM OF Miss F. F.
DATE January 6th 1930.
Case No. 36.
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
LINE AT A INDICATES READING OF AUDIOMETER WHEN TESTING A PERSON OF NORMAL HEARING WITH THE D-80904 BONE CONDUCTION RECEIVER
A
Right
Left
air- 7 sec.
10 sec.
256 D.V.
bone- 9 sec.
8 sec.
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
16 32 64 128 256 512 1024 2048 4096 8192 16384 32768
C1 C c c1 c2 c3 c4 c5 c6 c7 c8

Diet and pancreatic extract prescribed.


AUDIOGRAM OF Mrs. F. F.
DATE June 9th 1930.
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
LINE AT A INDICATES READING OF AUDIOMETER WHEN TESTING A PERSON OF NORMAL HEARING WITH THE D-80904 BONE CONDUCTION RECEIVER
A
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
16 32 64 128 256 512 1024 2048 4096 8192 16384 32768
C1 C c c1 c2 c3 c4 c5 c6 c7 c8

COMPILATION OF CASES.

Case No.	Name	Age	Age of Onset	Heredity	Other conditions	Duodenal Contents: Blood sugar cc.	Trypsin cc.	Amylopsin cc.	Steapsin mgms.
1	Miss T. L.	41	36	None	Pituitary dysfunction	79.8	100.	50.	296.
2	Mr. S. B.	45	43	None	Gall stones	133.3	100.	83.	408.
3	Mr. M. C.	27	21	None	Pericholecystic adhesions	72.	50.	100.	398.
4	Miss M. P.	26	21	Sister deaf	Amenorrhea	90.	34.	71.	704.
5	Mrs. G. V.	45	31	None	Typhoid at 23 yrs.; indigestion	105.3	25.	125.	224.
6	Mrs. R. S.	47	35	None	Followed pneumonia	112.	25.	125.	333.5
7	Mr. P. H.	49	46	None	Gall stones	108.8	25.	40.	704.
8	Mrs. F. K.	49	48	None	Cataracts, facial neuralgia	72.	25.	62.	840.
9	Mr. H. S.	40	39	None	Pericholecystic adhesions	82.	17.	33.	634.
10	Mrs. L. M.	26	24	Mother deaf	None	101.3	10.	25.	464.
11	Mrs. A. A.	38	37	None	Intestinal indigestion; Enlarged thyroid	111.	10.	50.	182.
12	Mrs. H. T.	54	49	None	None	116.	14.	10.	155.
13	Miss A. P.	21	20	None	Belching	140.	30.	5.	70.
14	Mr. G. S.	46	44	Brother deaf	Diarrhea; fullness after eating	86.	14.	7.	704.
15	Miss P. L.	24	20	None	Fullness after eating	82.	12.	5.	732.
16	Mr. H. D.	32	30	None	Belching 2 to 3 years. Cannot eat fats	94.	11.	83.	40.
17	Miss P. G.	36	15	Father deaf	Diarrhea for 21 yrs.	131.6	10.	0.	761.
18	Mr. A. V.	66	63	None	Pancreatitis	69.	10.	trace	0.
19	Miss M. B.	37	22	None	None	100.	8.3	165.	21.12
20	Miss H. S.	27	25	None	Tremor; enlarged thyroid	133.	5.	210.	690.

COMPILATION OF CASES—(Continued).

Case No.	Name	Age	Age of Onset	Heredity	Other conditions	Blood sugar	Duodenal Contents		
							Trypsin	Amylopsin	Steapsin
						cc.	cc.	cc.	mgms.
21	Mrs. F. S.	38	30	None	Enlarged thyroid; vomits; heartburn	82.	5.	107.	396.
22	Miss I. P.	30	20	Sister deaf	Headaches	103.3	5.	80.	185.2
23	Mr. H. G.	20	19	None	Heartburn	118.6	4.	180.	650.
24	Miss R. D.	50	44	Father deaf	Pathological gall bladder	125.	3.3	21.	111.
25	Mr. H. F.	48	47	None	None	82.	3.	135.	380.
26	Mr. I. S.	36	28	Maternal aunt deaf	Chronic Ethmoiditis	132.4	2.6	62.	272.
27	Mrs. G. S.	50	49	None	None	96.	2.5	125.	591.
28	Mr. J. C.	53	43	3 deaf paternal side	Intestinal indigestion	81.	2.5	30.	394.
29	Mr. G. Mc.	48	38	Mother and brother deaf	Duodenal ulcer, O.M.P.C. left	103.3	2.5	83.3	108.
30	Mr. M. G.	43	42	None	Biliary dysfunction	82.4	2.5	50.	0.
31	Mrs. S. R.	39	30	None	Influenza 9 yrs. ago; Cholecystectomy 2 yrs. ago	90.	2.5	61.	286.
32	Miss E. G.	19	17	None	Attacks of nausea	74.	2.5	20.	242.
33	Mrs. B. G.	33	28	None	Belching; stomach trouble 8 yrs. ago	103.	2.	60.	194.
34	Mr. S. L.	74	69	Mother deaf	None	125.	2.	1.8	183.
35	Mrs K. K.	49	46	Daughter deaf	Aniso arthritis	111.3	2.	50.	1028.
36	Miss F. F.	25	21	None	Headache; pathological gall bladder	82.	1.	62.5	220.
37	Mrs. F. L.	37	28	Mother and sister deaf	Enlarged thyroid; flatulant	95.2	1.	1.	549.
38	Miss P. W.	28	26	None	Vasomotor rhinitis	100.	0.	5.	408.32

Minimum requirements for digestion consistent with good health: Trypsin, 10—; Amylopsin, 20—; Steapsin, 50—.

CONCLUSIONS.

Pancreatic insufficiency is encountered in a large percentage of cases of nonsuppurative deafness. Deafness is often encountered in patients suffering with gall bladder disease. Pancreatic insufficiency and hepatic dysfunction are at times associated. Pancreatic insufficiency does not always coexist with hepatic dysfunction or biliary disease. That deafness is definitely due to the pancreatic insufficiency is demonstrated by the fact that under suitable replacement therapy the hearing improves, and when the treatment is discontinued the hearing deteriorates. No matter how profound the deafness, if it is due to pancreatic insufficiency it can be improved.

I desire to express my gratitude to the friend and counselor who has given me financial aid to pursue these investigations.

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DISCUSSION.

Dr. Samuel J. Kopetzky, New York City: This work of Gottlieb is presented to you as a piece of serious research work, a series of observations in a type of deafness with the results as given in his paper in the Presession Volume. A certain degree of corroboration is furnished in the communication of Dr. Drury, another reliable observer, but who observed his cases from an entirely different angle than the approach taken by Dr. Gottlieb.

Stacy Guild of Johns Hopkins University has recently shown the relationship in cases of deafness upon examination histologically and by the audiometer, of the cochlea to the functioning membrane. He shows audiometer curves that are similar to those shown by Dr. Gottlieb, and proves nerve destruction in special areas. *It is illogical to conclude that the giving of any pancreatic ferment would replace tissue whose function has been destroyed by fibrosis or cicatrization,* but the cases submitted by

Dr. Gottlieb are those of a much earlier stage—they are all early cases. All deafness does not fall into this one group, nor in the group of deficiency diseases, but all types have a common symptom—a degree of deafness, and this work at this stage of its development is presented to you because of the rather hopeless character of our therapy for these unfortunates later in the course of their lesions, because when a case then comes within the category, within the limits of those submitted by Dr. Gottlieb, perhaps it is not totally hopeless, but with this test of the pancreatic ferments at least we will be able to place the case in one of the groups and thereby give it something that may help.

I reiterate, the work is presented at this stage because enough experience has been accumulated to warrant your trying it and making your own experience. I distinctly reiterate that this is no cure for deafness. It is only an observation upon a small number of cases. The work should be checked by other observers. At present it is only a preliminary presentation.

Dr. E. M. Josephson, New York City: We should hail this paper as one of the group now growing in number that treat of chronic progressive deafness as a disease of a human organism rather than as a disease of an isolated organ.

When we begin to discuss the physiology of the external secretions we enter into fields unknown and unexplored. We find that research workers in the field of gastroenterology, including McClure, have found it impossible to establish normal figures for either the timing sequence, the rate or the quantity of pancreatic enzyme secretion, for it is extremely difficult to establish controls in this work and the variations are from zero to as high as 600 times the low figure, in the course of prolonged examination of a single case, according to McClure. He points out that the very introduction of the duodenal tube introduces a margin of error, as does the consequent intermittent passing of gastric secretions into the duodenum about the tube. It has been fairly conclusively proved, however, that the latent period is at least 45 minutes instead of the 15 minutes used by Dr. Gottlieb. According to the work of Dr. Hirschberg, published in the 1928 Archiv. f. Verdauungskrankheiten, the magnesium sulphate solution which Dr. Gottlieb introduces into the duodenum produces an enormous error both by alteration of the nature of the enzymes and by the inhibitory action which the magnesium sulphate has upon the enzyme action.

Dr. Gottlieb appreciates the unreliability of his method when he states: "We are aware that the methods used do not produce consistent results and accurate figures because of certain chemical and biologic facts." We fully agree with him. The margin of error in his tests for pancreatic enzymes are so large as to make any conclusions based upon them utterly unreliable.

Were Dr. Gottlieb's data free of these objections there would still be considerable doubt as to the etiologic relationship between pancreatic enzyme activity and deafness. Dr. Gottlieb points out that not all cases of pancreatic disease are deaf; also that many cases of chronic progressive deafness have what he interprets as normal amounts of enzymes, and that

a large proportion of the cases deficient in enzymes in his estimate do not respond even to his inconvenient mode of therapy.

As for the results which he obtains, it can hardly be agreed, however, that the amount of temporary hearing change which he secures is any greater than the normal variations of hearing in these cases. We all know that on some days the patients will come into our offices complaining bitterly, and on other days they will state that their hearing is improved, without any treatment. Some of the factors which influence the variations of hearing which are so characteristic of chronic progressive deafness are the following:

1. Variations in barometric pressure. A normal climatic change of one-half inch of mercury often gives over ten decibels change in hearing.

2. Variations in temperature may give variations of twenty decibels or more. Irrigations of hot water and hot air have been used with good temporary results by Pugnat and myself.

3. Variations in psychic state may account for as much as forty decibels change in hearing; which introduces a topic which I can only hurriedly allude to here, the anxiety neurosis and sympathicotonia which characterizes so many cases of chronic progressive deafness.

4. Posture changes and fatigue, which result in changes in hearing as well as in tinnitus. I might state that my detailed studies on tinnitus, by the use of masking effects, which enable determination of both pitch and intensity, disagree with Dr. Gottlieb's conclusions.

5. Cardiac insufficiency induces changes in hearing of the type attributed by Bunch to old age. These clear up under digitalization and other therapy, with hearing changes ranging to thirty decibels or more, and not infrequently restoration to normal. I have been able to observe directly with the ear microscope the improvement of the ear drum circulation under digitalis.

As indicative of the potential variability of hearing in these cases, I will mention a condition which will shortly be described in the literature. There has hitherto been unrecognized a type of deafness due to reflex spasm of the muscles of the auditory accommodative mechanism of the middle ear, which is not infrequently found in chronic progressive deafness and which accounts for failure of relief of drum retraction in the absence of eustachian tube blockage and adhesions. It is associated with deafness particularly, of the low tones, often as high as thirty decibels in extent. Some or all of the defect is remedied rapidly by galvanization in these cases. Miss A. A., case 11, represents the type of audiometric curve which is found in reflex spastic deafness.

Digestive disturbances, usually constipation, are found in a high percentage of the cases of c. p. d. We all know that the feces of a putrefactive constipation are more toxic than the normal, and there is no reason why they should not be more toxic in their effect on the guinea pig, which Dr. Gottlieb has shown to be true. When the constipation is relieved, the hearing usually improves often fully as much as in the cases here presented. I have found that administration of digest ferments, such as enteric pills of trypsin, papaine and caroid and others may or may not result in relief of constipation. My results with enzyme therapy

have been no better than is obtainable by treatment of the constipation.

Evidence is accumulating that the digestive disturbances of chronic progressive deafness as well as the inciting factor in the deafness are both the effect of disturbance of the nervous system, central, sympathetic and parasympathetic; that is sympathocotonia and vasotonia that predominates. There is reason to believe that treatment or ablation of the superior sympathetic and carotid nerve ganglia may give excellent results in these cases.

Case No. 27 could readily be suspected to be of cerebral origin. May I ask Dr. Gottlieb what were the blood pressure and the neurologic findings in this case? Also whether he has run any control cases under his regime of enema and mild catharsis without enzyme? It has been my experience that the results are fully as satisfactory as those which he describes.

DR. MARK J. GOTTLIEB, closing: In regard to what Dr. Kopetzky said about the destruction of nerve tissues in progressive deafness, there is no doubt from my experience in treating some of these cases that there is some improvement. Beyond that improvement, however, it is utterly impossible to obtain a more favorable result. It is likely that fibrous tissue has replaced the essential elements which convey hearing impulses to the brain, and it is possible also that the work which Dr. Kopetzky mentioned will substantiate that as time goes on.

Regarding what Dr. Josephson has said, I will not say anything about the parts of his discussion which have nothing to do with my presentation. That would involve too much and as it is not relevant it might raise a great deal of question. However, I do want to say this, that one must have more than a theoretical acquaintance with the subject before one enters into a discussion such as Dr. Josephson presented. For instance, you cannot give medicines such as enzymes by mouth indiscriminately and expect to get results. You must know first whether the patient's deafness is due to pancreatic insufficiency. There must be a differential diagnosis made in every case. Furthermore, the preparations which Dr. Josephson mentioned are utterly useless. Pancreatic extract must be given in large quantities and it must be powerful. Otherwise it is of no use. Weather conditions make no difference in the hearing tests of these cases.

Regarding the case about which Dr. Josephson raises the question that it may be one of brain tumor, I would say that every known clinical and laboratory test was done, and these disclosed no evidence of intracranial neoplasm.

THE BESREDKA THEORY OF IMMUNITY AND ITS POSSIBLE APPLICATION IN OTOLARYNGOLOGY.

FRANK J. NOVAK, JR., M. D.,

CHICAGO.

The title of this paper originally included the relation of the reticuloendothelial system and the Besredka theory. Upon further consideration it was thought more expedient to contract the subject, limiting it to a presentation of the theory alone with some of its implications, reserving consideration of the extended subject for another paper or, if it is desired, to consider it briefly in the closing discussion.

A new concept of immunity was presented by Professor A. Besredka of the Pasteur Institute about three years ago. Excepting the men who are actively engaged in immunologic studies there has been little, if any, interest in Besredka's work in this country. Clinical application and investigation of this new theory have been neglected almost completely except in France and in some of the other European countries. In America, Besredka's revolutionary idea of immunity has engaged the academic interest of such investigators as Cannon of the University of Chicago, and Frederick P. Gay of Columbia University.

My purpose in presenting this paper is to try to stimulate clinical interest in this new conception of the mechanism of the immune reaction. I do not propose to give my clinical experience because it is too limited to bear any weight.

The early bacteriologists predicted that the future physician would be an immunologist. Today the average otolaryngologist is very skeptical about the value of the various immunologic practices which are available in the management of the ordinary head cold. Vaccine therapy for infections of the upper respiratory tract is generally regarded as of doubtful value. Besredka proposes a modification in the preparation of vaccine and gives a radically new method of administering it. I shall try to present a brief outline of this work without presuming to endorse or to criticise it.

The first in the list (chronologically) of the pathogenic bacteria was the anthrax bacillus. In the year 1850 Davaine observed

the presence of small filiform, nonmotile bodies in the blood of a sheep dead of anthrax. He believed that these "filiform infusoria" caused the disease, anthrax. He did an enormous amount of experimental work to prove that the anthrax bacillus was the specific cause of anthrax. His work, however, was not accepted until twenty-seven years later. In 1881 Pasteur made his memorable experiment on 24 sheep, 6 cows and one goat. These animals were vaccinated against anthrax. Then all of the animals were inoculated with a virulent culture of anthrax. None of the animals succumbed. Since that time it has been learned, however, that vaccination of the large animals produces solid immunity in only a relatively small percentage. The vaccination is not unattended by considerable danger to the life of the animal. Moreover, it is known to every laboratory worker that it is impossible to produce immunity in the small laboratory animals, particularly in the guinea pig. It is axiomatic that a guinea pig whi-h comes in contact with anthrax is already a dead guinea pig.

Besredka, studying anthrax, found that a guinea pig whose skin was desensitized by applying a wet compress of an anthrax filtrate to the roughly shaven skin of the belly could receive a dose of live, virulent anthrax bacilli with total impunity. In other words, by immunizing the skin of a guinea pig the whole animal becomes solidly immune to anthrax. The injection of either vaccine or filtrate into the blood, subcutaneously, intramuscularly, or giving it by mouth, fails to produce immunity to anthrax in the guinea pig, but injecting a dose of anthrax filtrate intradermally confers upon the animal a solid immunity. A wet compress appears to be as efficient as the injection, provided the skin is roughly shaved, producing a slight abrasion of the skin. In rabbits immunized by Besredka's method a dose of virulent organisms, three thousand times the minimum lethal dose, failed to kill the animal.

In order to prove that the skin of a guinea pig must be infected with the anthrax bacillus in order to produce the disease, the following experiment was done. Some method of introducing the organisms into the animal without contaminating the skin, had to be employed. Injection through the skin does not preclude the possibility of contamination of the skin with the needle. Plotz used glass, gelatine, and collodion capsules, which were filled with virulent cultures of anthrax bacilli. These were imbedded subcutaneously through a small incision which was

allowed to heal. These capsules were then broken, liberating the live cultures in the subcutaneous tissues. The animal suffered no harm. The organisms became "inoffensive when they came in contact with the subcutaneous tissue, and isolated from the skin." However, the organisms that remained in the unbroken capsules did not become attenuated even after six days.

Besredka believes that he has shown that in the guinea pig it is necessary to infect the skin with the anthrax bacillus in order to produce anthrax. Secondly, that the buccal, pharyngeal and gastrointestinal mucosa is impervious to this bacterium, provided the mucosa is not injured by a sharp object. In other words, the organism does not penetrate an intact mucosa. Anthrax then is a skin infection per se. All previous efforts to immunize a guinea pig to anthrax have been futile. Injection of the skin itself with a filtrate of an anthrax culture produces a general solid immunity.

This method of immunization received a searching trial in Syria in 1923. The French cavalry patrolling the wheat fields suffered an alarming loss of horses from anthrax. The Pasteur method of vaccination proved dangerous and was given up. Upon using intradermal vaccination of the filtrates the average mortality among the animals was reduced about twenty times. Altogether about nine thousand horses and mules were vaccinated. The director of the veterinary service called this "a success without precedent in the history in antianthrax vaccination in horses."

In 1923 the commission on epidemics of the League of Nations was confronted with an epidemic of bacillary dysentery among the thirty thousand refugees in Greece. The tissue involved being the gastrointestinal mucosa, an attempt was made at vaccination of this tissue by Besredka's method, using a filtrate of cultures of the Shiga Bacillus. The filtrate was ingested. In one group of seven hundred the epidemic stopped "abruptly and completely," upon vaccination of the entire group. Three hundred and forty individuals arrived at Kokinia, where an epidemic was raging. The new arrivals were vaccinated and although they drank the same water as the rest of the camp, among the vaccinated individuals no dysentery appeared. At the camp of Kokinia four hundred cases of dysentery occurred among 4800 refugees. Two-thirds of the 4800 were vaccinated. In this vaccinated group the epidemic stopped immediately and

completely. In the unvaccinated group the epidemic continued for many months.

In a paper entitled "Intradermal Injections of Gonococcal Bouillon Filtrate," Corbus and O'Connor say, "in the treatment of eight women with gonorrheal endocervicitis, four of whom originally presented themselves with salpingitis,—all the tubal symptoms subsided promptly after their first injection and three were gonococcus-free after four weeks. In three young girls with vulvovaginitis of specific origin the results were equally promising. Thirty-seven men have been or are under controlled treatment by this method. Nine are apparently cured after no other type of treatment, systemic or local, but must be kept under observation for several more months to satisfy the most rigid requirements.

Now turning to the matter of the staphylococcus and the streptococcus we find that these organisms have a very low antigenic value. It accounts for the inefficacy of staphylococcus and streptococcus vaccines. Comparing these two organisms with the anthrax bacillus Besredka says "they have, however, one characteristic in common, and that is a negative one; namely, their inability to serve as antigens. Besredka says that a filtrate of a streptococcus and staphylococcus culture, which has been heated will, when injected into the skin of an animal or man, produce immunity which is specific against these two organisms. He cited much experimental work and refers to a considerable amount of clinical work in support of his statement.

Concerning the theoretical basis for local immunization Besredka quotes Pasteur on scientific theories in general when he refers to them as "the illusions of the experimentor, which serve as a guide, and which gradually fade as we travel along the road." In the first place the filtrate consists of the medium in which the organisms have been grown; usually broth or broth serum. The living organisms are filtered out. Now if the filtrate is reinnoculated the bacteria will grow but sparingly. After the second filtration a further effort to innoculate the medium fails. The organisms remain viable but do not grow. This second filtrate contains two substances. One is toxic and thermolabile, and the other is nontoxic, and thermostabile. The filtrate is now heated at 100° Centigrade. This heating destroys the first or toxic thermolabile substance, which Besredka calls virus. The remaining, thermostabile substance, he calls anti-

virus. The filtrate is now ready for use either as wet dressing or for intradermal injection.

There are two general theories of immunity, the humoral theory, and the cellular theory. It is clearly not within the province of this paper to enter upon a discussion of these theories except as the latter differs from the former in a few salient particulars. Besredka believes that antibodies have but a subordinate part in the immune reaction. They are the digestion products of the interaction between bacteria and phagocytes. When a tissue like the skin reacts with an organism like the anthrax bacillus it does so because the skin cells are sensitive to that particular organism, or some of its products. The application of the filtrate or antivirus, as Besredka calls it, to the skin satisfies this affinity. To put it in other words it desensitizes the normally receptive cells of the skin. Now following this desensitization, when a living culture of anthrax is inoculated into the skin, the cells being no longer sensitive, there is no reaction between the invader and the host and consequently there is no reaction of inflammation. The anthrax bacillus is forced into the role of a saprophite and is promptly phagocytosed largely by the macrophages of the reticuloendothelial system, as Cannon of the University of Chicago has shown. The immune bodies which are found in the blood are considered by Besredka merely as by-products of the immune reaction. He cites the fact that in certain diseases to which the individual succumbs a large quantity of immune bodies are found, and again in other diseases which the individual survives there is an almost total absence of immune bodies in the blood. Therefore, if the mechanism of immunity is due to the formation of immune bodies the two observations mentioned above are inexplicable. It more easily satisfies logical requirements to assume as Besredka does that the immune bodies are but an incidental product, and that their presence or absence, increase or decrease, does not in any way indicate the presence of or the degree of immunity of an individual.

CONCLUSIONS.

The experience of laryngologists with the various vaccines in the treatment of infections of the upper respiratory tract has been unsatisfactory. At the Pasteur Institute under Besredka a new conception of immunity has been worked out. It has had an extensive trial in Europe, both in the laboratory and in

the clinic. It deserves extensive clinical investigation here, and I do not know of a more suitable body for that purpose than the Academy. I recommend a careful reading of two books by Besredka: "Local Immunization—Specific Dressings" and "Immunity and Infectious Disease," both published by the Williams and Wilkins Company.

30 N. Michigan Avenue.

DISCUSSION.

Dr. Austin A. Hayden, Chicago: I think the Academy is to be congratulated on having this important subject brought to it in such a concise and skillful fashion. As Dr. Novak has said, the experience of otologists in general has not been very favorable to various vaccine therapies. The very interesting clinical data that he has cited in regard to anthrax, its being purely and exclusively a skin disease, recalls to our mind the fact that other organisms have specific infectious properties for specific tissues. The gonococcus, for instance, attacks only the mucosa of the urogenital tract and the eye, but unlike anthrax, its virulence is not in any way attenuated, at least no experimental work has been done on the attenuation of this organism as has been done in the case of anthrax. I presume the fact that the skin does react under circumstances and under other circumstances does not, is due to the slowness of the infection, or to the absence of infectious products, so that scarifying the skin may produce a localized immunity which protects the organ from actual infection by the organism.

This field certain presents a very interesting speculative future for this type of therapy, and I presume it is not a far stretch of the imagination to see the application of this therapy to the ordinary head colds and sinus infections and, as Dr. Novak told me yesterday, perhaps to infections of the lungs and to intra-abdominal infections, with the possibility of immunizing the peritoneum and the pleural cavity. If any such thing does occur, the Academy will have been particularly fortunate in having one of the very first presentations of the subject made before this body.

Never having used this method I have little personal knowledge of the procedure, but Dr. Novak's presentation of the question interests me profoundly.

Dr. Joseph C. Beck, Chicago: I am glad to hear Dr. Novak present this subject. I have had some experience with it and I think it is going to prove of considerable value. As I look back a number of years ago I remember that Dr. Harold Hays mentioned something about the way he used dry bacteria locally. It seems that it is only right that we should remember that such a method has previously been used by a member of this Academy.

But I know so little about the subject, and there is a man here who knows so much, that I would like to hear from him—Prof. Lloyd Arnold, of the University of Illinois.

PROF. LLOYD ARNOLD, University of Illinois: I think I can probably conserve time by asking myself a few questions that you probably have asked yourselves, and let that count for discussion.

First, what is the Besredka antivirus? How does it differ from the vaccines with which we are familiar?

Second, what is the relationship between this Besredka antivirus and d'Herelle's bacteriophage? Both substances have been applied to the upper respiratory tract under various conditions.

First, the Besredka antivirus is practically a five to eight-day old culture. It is passed through a bacterial filter to remove all of the viable bacteria. There are a great many things taking place in old culture. Some of the bacteria that are destroyed are dissolved, some die because of by-products of metabolism or acidity of the culture. Besredka does not use sugar so the cultures remain acid. The most of them undergo destruction in a period of four or five days.

d'Herelle's bacteriophage is a much more complicated substance. He considered he had discovered a substance which passed through the bacterial filter that was a virus. It caused the condition of bacteriuria. It was a pathologic virus that caused the dissolution or death of the bacteria.

As a result of our work for approximately eleven years we have concluded that this substance that dissolves the bacteria is a ferment. It breaks the cell and liberates bacterial proteins in solution. We do not know that the bacteriophage itself has any curative value whatever. We have been unable to separate bacteriophage, the lytic agent, from the bacterial proteins. The lytic agent is just a scientific curiosity.

The second question: What is the relationship between Besredka's antivirus and d'Herelle's bacteriophage? The two are close together; both are bacterial proteins. I think the clinical results are due to specific bacterial protein therapy. There is nothing new about it. Besredka's work on anthrax was very interesting, it was the best piece of work he did. The work upon the staphylococcus and streptococcus is far from convincing. We are now beginning to realize that we have other types of socalled vaccines than those we usually produce by growing cultures and destroying the reproductive power of the bacteria by heat or some chemical agent. We now have two other means of producing substances that render soluble bacterial proteins. One is by autolysis, and the other is the use of d'Herelle's principle, using a ferment and growing a culture. The culture will be dissolved in six to eight hours. Then we have a bacterial protein suspension of young bacteria. The only difference between these two substances is that the Besredka method produces autolysis causing a liberation of ferment inside the bacteria itself. With the d'Herelle method we have a ferment that dissolves the bacteria during their growing stage.

The application of these to the mucous membrane or skin presents many points of interest. We are now finding that these dissolved bacterial proteins can pass through the skin and also through the mucous membrane surface. I think that is a step forward and undoubtedly presents some phases that have great possibilities. From a scientific standpoint

we have been working with the two substances a good deal. Our old type of vaccine probably was wrong in that we changed the proteins by heating it or adding various things to it. Here we have two types of protein from bacteria, one by Besredka on autogenous lines, and the other by d'Herelle on ferment lines. We do not add anything to either of these. The two will possibly remain what we might call ideal vaccines.

Dr. A. W. Morse, Butte, Montana: Besredka's work was brought to my attention by Dr. John X. Newman, bacteriologist, of Butte, who prepared for me an autogenous antivirus according to Besredka for three or four chronic, resistant cases. Two cases which showed the most improvement I cannot report here as they were chronic recurring conjunctivitis with blepharitis marginalis. The case I wish to report was one of double antrum infection of long standing. Adequate openings under the lower turbinates had been made, and for two years there were times of intensive local treatment, and periods of no treatment. There was always some discharge, at times profuse. The infection was mixed, staphylococcus and streptococcus predominating. Autogenous antivirus according to Besredka was made and used locally for six months, first daily, then every second day, and then at intervals. It did more than any other treatment to control the discharge; it shortened the exacerbations, but it did not affect a cure. Because of beginning bronchitis and choroiditis it was necessary to do a double Caldwell-Luc operation with entire removal of the infected mucous membrane.

Dr. Ralph A. Fenton, Portland, Oregon: It is to be hoped that Dr. Novak will continue his research toward the basic factor in immunity: the reticuloendothelial defense, which he suggested at the 1927 meeting.

Besredka's epochal contributions, so vital in the control of dysenteric diseases, have not thus far advanced from the stage of controversy respecting immunity to the streptococcus. Harold Hays' work, following Besredka's, in 1925, on the local use of mass cultures, has remained unstabilized, with certain elements of danger.

Surface exposure to vaccinating substance is of course impracticable within the nasal accessory sinuses, especially where bacteria have penetrated below the edematous surface. The bloodstream, and the leukocytes entering each remote capillary with its enveloping histocytes, must hold the key to this riddle. Some common factor in immunity—perhaps the socalled "reticulia" secured by Charlton of Pasadena from the omentum of the pig—certainly some factor excited by alien protein therapy as well as specific vaccines—awaits the further industry of the bacteriologist and of the philosophic clinician like Dr. Novak.

Dr. Frank J. Novak, closing: I wish to thank the gentlemen who discussed the paper.

Dr. Hayden mentioned the gonococcus. Dr. B. C. Corbus has done a great deal of work on the gonococcus and an antivirus filtrate and has reported twenty or twenty-five cases which he has observed in which he has secured brilliant clinical results.

Dr. Fenton mentioned the reticuloendothelial system, and that brings up immediately the crux of the whole situation. What is the mechanism

of this thing, what does the socalled virus form from, what are the mechanics of it?

In the first place, the reticuloendothelial cells should be called histocytes because they are not endothelial in origin, and are concentrated in the greatest bulk in the spleen, liver, and the lymph glands. They are also observable in the skin and around the blood vessels. The histocytes are derived from the lymphocytes. Maximow shows that the development of the histocyte is always a late condition. First we have in the germinal center of the lymph node, the lymphocyte. Later these lymphocytes migrate to the periphery of the germinal zone and become the small lymphocytes that we see. These small lymphocytes under proper stimulation develop into monocytes, and monocytes under proper stimulation develop into histocytes. Of course there is some controversy whether this chain of development is an actual fact. The histocyte is a fixed cell in the tissue, but it is capable of migrating. It may be anchored in its place, but under proper stimulation it may travel outside the tissue in which it is developed. It contains a great deal of cytoplasm, has a large nucleus, and has the capacity of ingesting foreign substances. It will ingest India ink or almost any foreign substance when it is functioning in its role as a macrophage. This macrophage which is capable of functioning in this conception of Besredka is the active agent in the immune reaction. These cells take up the organisms which get into the tissues which have been desensitized by the antivirus, and these cells carry out their function of ingesting the organisms which have become practically inert and destroying them. Then the product of that destruction—it is an enzyme—is the antibody, the agglutinin, the hemolysin, etc. Cannon, of the University of Chicago, has shown that by blocking the endothelial system by his method of injecting India ink and studying the cells, formation of specific antibodies is inhibited.

AGRANULOCYTIC ANGINA.

R. E. Windham, M. D.,

San Angelo, Texas.

The apparently increasing appearance of sporadic cases of agranulocytic angina seems of sufficient importance that all angles and new phases of this most baffling condition or disease should be brought up for discussion, especially as its prognosis is so unfavorable.

Schultz and Reye of Germany first brought this condition to our attention in 1922, giving it the name of agranulocytic angina and believing it to be a true clinical entity, while others have called it merely a syndrome.

Dr. Beatrice Lovett of Chicago, who seems to have recorded the first case in America, says that although no definite etiology has been found and the pathogenesis is as yet unexplained, the great similarity of symptoms of all cases, the blood picture and the course indicate that agranulocytic angina is a true clinical entity.

A striking feature of this disease is its prevalence in women of middle age, there being in some sixty reported cases only about a dozen cases in men and children combined; the vast majority of these were reported from Germany.

Another striking feature is its high mortality. Of the statistics collected by Kastlin there were three recoveries in the first forty-three reported cases, these being in Germany, my first recovery being the first recorded in America.

Let us pass in review the differential diagnosis of the diseases occurring in the oral cavity. We may safely exclude all the usual forms of stomatitis, catarrhal, aphthous, ulcerative, parasitic and membranous stomatitis and foot and mouth disease, or epidemic stomatitis, and consider for comparison or possible relationship gangrenous stomatitis, or noma, idiopathic gangrenous stomatitis, as classified and defined by Kraus, and agranulocytic angina, as defined by Schultz and Reye.

Let us pause for a moment for some comparative analyses of the three last mentioned conditions. Noma, or gangrenous stomatitis, we are told in the German medical literature, is a disease occurring almost exclusively in children under special conditions and is believed to be characterized by its special localization in the cheek; its typical course with few exceptions ends fatally.

The age given is usually two to five years and almost always during convalescence from an acute febrile process in unusually debilitated children, although atypical cases are admitted to occur in adults.

This condition practically always involves the cheek, often producing perforation. The Russian authors, who are very familiar with noma on account of its frequent occurrence in their practices, propose for this malady the name of gangrene around the angle of the mouth.

Its bacteriology, like the others, is unknown and it does not produce agranulocytosis.

Idiopathic gangrenous stomatitis, as classified by Kraus, which in his opinion though generally rare, occurs in vigorous adults.

The disease, as described by him, is marked by three characteristics: Primary necrosis, irresistible progress of the destructive process, total absence of any reactive inflammation between the vital and necrotic tissue.

The character of the lesions, their location, the fetid odor, are not unlike agranulocytic angina, though the marked agranulocytosis is absent.

Its bacteriology is also unknown, though Perthes and others believe a mycelial fungus of the streptothrix family to be the exciting cause of noma and idiopathic gangrenous stomatitis.

In agranulocytic angina the onset in all cases is similar and sudden, with a severe sore throat and fever. The throat shows ulcers and exudate, particularly on the tonsils, resembling a dirty diphtheritic exudate, and in most cases on a dark necrotic base. Next in frequency are the gums and hard palate, and there are reported cases of the genitalia showing similar appearing lesions.

An infectious and gangrenous process common to all of them is predominating.

The cardinal symptoms of this process are the necrotic disintegration of tissue; the malignant course is indicated by the

irresistible necrobiotic process which gradually involves new areas of tissue. Histologically the leucocytic reaction so commonly present in any inflammation is absent and the defensive power of the tissue seems totally impeded.

Moore and Wieder of Philadelphia state that skin and intestinal lesions have been found and that the most characteristic anatomic change is found in the bone marrow, which appears to be the seat of the disease.

They state that where there is opportunity to examine the bone marrow, the absence from this of neutrophiles, myelocytes and myeloblasts and the presence of megakaryocytes, erythrocytes and erythroblasts are conclusive evidence of agranulocytic angina. Others have tried to prove the condition a systemic disease, with primary foci possibly in the liver, and the angina of secondary nature.

It is my belief that the anginal manifestations are primary and the toxemia secondary.

The lesions all begin as whitish or gray areas, turning to a dirty gray, then to a blackish gray on a blackish, necrotic, gangrenous base, with a very offensive odor. The odor, I believe, is almost diagnostic and pathognomonic.

Some report cases as being icteric, while Zadek and others report cases showing a thrombopenia and marked tendency to hemorrhage.

My first and fatal case was icteric and showed no bleeding tendency, but the three subsequent cases, all of whom recovered, showed no icteric condition, but all had bleeding tendencies.

The disease is self-limited to mucous membranes, destroying them down to the muscle but never going beyond them, the gum and palate tissue being completely destroyed down to the bone; and when the tonsil is involved a complete tonsillectomy by slough occurs, thus showing a susceptibility of mucous membrane and lymphoid tissue.

Bacteriologically the question is still unsettled, but from the nature of the condition and the apparent prevalence of bacillus pyocyaneus in so many cases, there seems no question but that it may be one of the causative organisms, together with some anaerobic organism, obligatory or facultative. Other organisms have been recovered, as streptococci, staphylococci, diphtheria

bacilli and Vincent's spirillæ but have been considered a coincidence.

Perthes mentions a mycelial fungus of the streptothrix family, while others consider a fusiform bacillus or a spirillum as the guilty organisms.

Castellani claims the constant presence of two bacilli in fetor oris of tonsillar origin, for which he proposes the names bacillus colofoetidus and bacillus alkalofoetidus, but it yet remains to be proven if they are the organisms of agranulocytosis.

The clinical picture, other than the lesions of the mouth and throat, temperature and blood picture, is of no consequence. No other evidence of disease is found, even to a mild cervical gland involvement.

Pathologically there seems to be but little information found at autopsy. The liver, spleen and kidneys are unchanged except for slight enlargement; a parenchymatous degeneration and some passive congestion of the liver. There is normal red bone marrow. These findings, I believe, disprove the theory that the condition is systemic in origin with a local manifestation in the throat and support the theory that it is definitely a local clinical entity, later developing a septicemia.

The blood picture seems very characteristic if not pathognomonic, there being a moderate to severe leucopenia, the total leucocytes going down to a few hundred and the polymorphonuclear leucocytes very low, to almost zero in given cases, while the mononuclears are greatly increased, up to 90 per cent and more; the hemoglobin percentage remains about normal and there is no anemia.

Histologically there is not much information to be gained. The liver shows only areas of diffuse perivascular lymphocytic infiltration. In the spleen there is marked development of sinus endothelium at the expense of the lymphatic tissue, again proving a susceptibility of lymphoid tissue to the ravages of the disease. The kidneys showed numerous bacterial emboli through the cortex and some in the medullary substance; the absence of inflammatory changes was very striking.

The local character of the tissue attacked by the malignant necrosis and the missing defensive reaction of the surrounding tissue prove that the protective forces of the whole organism are failing.

COMMENT.

Because agranulocytic angina attacks the throat it may be confused with diphtheria or Vincent's angina, or one of the several stomatites.

There is never any appearance of the condition in the nose or the nasopharynx, which is evidence that it does not thrive where there is free circulation of oxygen, but does where oxygen is somewhat excluded, circulation less active and the tissues with lower resistance.

The organism seems to be definitely anaerobic, and the treatment should be instituted with this in mind to meet the offender in its own lair.

The disease is not common in children, possibly due to their vigorous circulation and the heavy oxygen content of their blood, but is prevalent in middle aged women when their circulation is naturally slowed down, when they are undergoing endocrine disturbances and the tissues are poorly supplied with oxygen.

The disease is a local one, seldom producing septicemia, as all anaerobic organisms avoid the blood stream because of its oxygen carrying power. The tissues of invasion are areas where free circulation of air is restricted, as in the oral cavity and the vaginal tissues.

TREATMENT.

Looking at the blood picture, I decided to use neoarsphenamin as a blood stream purifier and blood builder, as we know arsenic is one of our best drugs for this twofold purpose.

Metaphen was also selected as a blood stream purifier, as it is believed to be far superior to other preparations for this purpose. These two drugs were given intravenously on alternate days for three or four doses.

As a local application I wanted something strong in germicidal and penetrating power, and something that would carry free oxygen deep into the tissues. For this weapon I went into the laboratory and started mixing various substances, acids and chemicals.

The mixture of chromium trioxid and hydrogen peroxid proved to liberate the highest content of free oxygen and at the same time penetrated the tissues, carrying free oxygen into the affected tissues.

The areas are cleansed of all necrotic tissue possible; then chromium trioxid crystals are applied well into the crevices; these are picked up on a tightly wound cotton applicator dampened with water and gently massaged in well; then a loosely wound fluffy applicator is dipped into hydrogen peroxid (fresh) and massaged over the chromium trioxid application, which, when the two combine, liberates a large amount of oxygen. The chromium trioxid unites with the saliva to form chromic acid (orange color). The hydrogen peroxid then oxidizes the chromic acid to perchromic acid (blue). Then the two mutually decompose, liberating nascent oxygen, producing a brown color. This last item is also specific for Vincent's angina in one to three treatments, at twenty-hour intervals.

An initial large dose of diphtheria antitoxin was given, both for possible associated infection and for the protein effect of the serum.

A strong mouth wash, either metaphen or hexylresorcinol, was given, to be preceded each time by hydrogen peroxid.

CASE HISTORIES.

Case 1.—Mr. B., age 62, a laborer, first seen on June 28, 1928, and admitted to Longview Memorial Hospital, complaining of general malaise and a very sore mouth and throat. He gave a history of having a number of teeth pulled twenty-five days previously.

Examination showed the teeth all out and a dark gray discharging gangrenous condition along the upper gum lines extending to the middle of the hard palate. The tongue was badly coated and there was a very foul odor. The right side of the face was slightly swollen and tender. The blood pressure was 148 over 68, the temperature was 103.4, pulse 120 and respiration 20 on admission. The physical examination was otherwise negative and the past history of no consequence.

This patient was icteric and somewhat anemic, the red blood cell count being 2,400,000 on admission and going down to 1,592,000 on July 6, three days before death.

On admission the total white cell count was 2,000. Hemoglobin was 45 per cent, polymorphonuclears 5 per cent, mononuclears 94 per cent, eosinophiles 1. On July 6th, the last blood count, the white count was 240, the hemoglobin 30 per cent, polymorphonuclears 4, and lymphocytes 95.

The blood culture was sterile. In smears staphylococci and streptococci were found. On culture bacillus pyocyaneus was found. Urine was negative throughout.

The patient developed a mild hypostatic pneumonia the last two days of life, and the temperature was 106 on the date of death.

Smears from his mouth rubbed into a rabbit's mouth produced no signs of infection, the rabbit being killed for safety.

The infection, which at all times was gangrenous and very foul, covered the patient's entire mouth before death.

Autopsy showed but little, as was previously mentioned.

The treatment of this case was more of a general nature, as little was known at that time as to what was best. The mouth was treated locally daily, but this patient did not get the detailed treatment as outlined and as was given in cases Nos. 2 to 4.

Case 2.—Mr. P., age 35, was admitted to the Longview Memorial Hospital on November 18, 1928, complaining for the last five or six days with general malaise, sore throat and aching.

A general physical examination was negative. Examination of the throat showed it somewhat red and swollen about the left tonsil, which was covered with a dirty gray exudate on a black base. There were about six small areas on the soft palate and the pharynx. On admission the temperature was 103, pulse 84, respiration 20; total white cells 7,900, polymorphonuclears 24 per cent, lymphocytes 75 per cent. During the illness the lowest white count was 1090 and polymorphonuclears 20. Urinalysis showed many urates and a few hyaline and granular casts.

On August 14, 1928, this patient had a 3 plus Wassermann reaction and received ten doses of neosalvarsan of 9 gm. each, between August 14th and October 15th, and seven doses of bismuth of 1 gm. each between October 14th and November 14th, before admission to the hospital on November 18th.

On November 20th, a puncture was made into the left peritonsillar area to relieve the pressure. On the 21st the left tonsil was lifted out as one necrotic piece. On the 22nd, the throat proper looked better, but on each side of the hard palate, opposite the first molars, there was a white area one inch square which involved about two-thirds of the hard palate. On the 23rd these areas were black and gangrenous and were lifted out, leaving the muscular coating. There was now an area outside the upper jaw opposite the second molar one-half by one inch and

inside and outside on the left lower jaw one-half by one inch on an area centering at the last molar, which were lifted out, leaving the bone bare. On this date I gave the family a bad prognosis, not believing he would recover. On the 25th, there were no new areas involved, and from then on there was gradual improvement, and the patient was discharged from the hospital on December 5th with the loss of much tissue and several areas of jaw bone exposed one-fourth by one inch, which areas continued to gradually heal over. The case received an initial dose of diphtheria antitoxin and on alternate days neosalvarsan and metaphen intravenously. If the lues and antiluetic treatment played any part in causing the disease, why did he get well, while still receiving the same medicine? Other treatment was as outlined. Treating the first case, I was at sea, but before having the second case I had done some thinking and felt better armed to tackle the second case.

Following the discharge of this patient from the hospital pieces of alveolar processes from both the upper and lower jaws, one-fourth by one inch, came away as sequestra from the areas where the soft tissues had previously sloughed away. The teeth here became very loose, necessitating their extraction. Blood culture was sterile; smears showed staphylococci, streptococci and Vincent's spirilla. Culture from mouth showed many streptococci and bacillus pyocyaneus; no Klebs-Loeffler bacilli. A piece of necrotic tissue from the patient's mouth was buried in a rabbit's mouth, and in five days showed a similar necrotic area. A culture from it showed bacillus pyocyaneus. The rabbit was killed and burned.

Case 3.—Miss C. H., a girl, 22 years of age, consulted me on September 27, 1929, complaining of an extremely sore throat of about a week's duration, with general aching and high temperature.

Examination showed a well developed, well nourished individual of healthy appearance. Past history was of no consequence.

The left tonsil was covered with a dirty gray membrane with black showing through; the right tonsil was black and gangrenous; there was a very foul odor and the surrounding tissues were red and inflamed.

On admission to the hospital the temperature was 103.5; the white count was 3,500, polymorphonuclear cells 10 per cent, and

mononuclears 90 per cent. The urinalysis was negative and findings otherwise were negative. The white count on the second day of hospitalization was 2,500, its lowest level; polymorphonuclears 8 per cent.

Also on the second day there were several small gangrenous areas on the hard palate posteriorly, but none subsequently.

The tonsils were completely sloughed away by the third day. The routine treatment was rigidly applied and the patient made an uneventful recovery, leaving the hospital on the seventh day.

Case 4.—Miss H. C., age 20 years, came to me in a very toxic condition on October 10, 1929. She gave a history of a progressive sore throat of a week's duration, becoming steadily worse under home treatment. The past history was negative, and on admission the temperature was 102.8, urinalysis negative, white count 4,200, polymorphonuclears 29 per cent and mononuclears 71 per cent. Both tonsils were covered with a grayish black formation with a very foul odor. The physical condition was otherwise negative.

On the second day the white count was 3,100 and polymorphonuclears 24 per cent, and on the third day the white count was 2,900 and polymorphonuclears 18 per cent, the lowest level reached. On the second day parts of both tonsils were removed and a gangrenous area on the hard palate posteriorly the size of a nickel was present. On the third day a complete tonsillectomy by sloughing occurred.

The routine treatment was rigidly instituted on admission and the patient made a good recovery, leaving the hospital on the sixth day.

CONCLUSIONS.

1. The disease is of the throat and other areas where the air is limited or excluded and is gangrenous in nature with a characteristic appearance and odor.

2. The offending organism is undoubtedly bacillus pyocyaneus plus some organism obligatorily or facultatively anaerobic in nature.

3. The disease has no definite limitations as to age, sex or social status.

4. It is becoming more prevalent and its prognosis is very unfavorable.

5. The type of organism, its anaerobic nature and the use of oxygen and chromium trioxid in its treatment have not been previously described.

6. So far as my cases and experience go, the disease is a clinical entity with the tonsil as the primary focus.

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DISCUSSION.

Dr. Frank R. Spencer, Boulder: Agranulocytic angina is characterized clinically as (1) a severe, septic sore throat, usually involving one or both tonsils, the pillars, soft palate and posterior pharyngeal wall; (2) probably caused by a variety of bacteria such as the streptococcus, staphylococcus, pyocyaneus, Klebs-Loeffler, Vincent's, etc.; (3) with severe general prostration and general sepsis, usually terminating fatally; and (4) with a low cell count for polymorphonuclear leucocytes and the mononuclears greatly increased. The disease may be a separate disease entity, but is probably only a severe type of sepsis beginning in the throat and closely allied to the types of streptococcic sore throat we have all seen and recognized for years. The streptococcus is the most likely cause of agranulocytic angina. The frequency of the disease in women of middle age is likely to be caused by their sedentary life and endocrine dysfunction. The disease resembles diphtheria, because it is localized in the throat, but the general toxemia may prove fatal without the use of antitoxin.

Foster has reported a case following the administration of neoarsphenamin. "The interesting features of this case were: the high, continuous fever, the prostration, the ulcerative stomatitis, the lack of a skin rash and hemorrhages, and the blood picture which showed no anemia or reduction of the platelets, but a definite and moderate leucopenia with a marked decrease of the polymorphonuclear neutrophil cells, and a relative increase of the lymphocytic group.

"Although stomatitis in this particular type of arsenic poisoning is not a constant finding, it is described rather frequently as noted by Dodd and Wilkinson who found stomatitis to be present in 11 of the 24 cases which they reviewed. In this same series of case reports, leucopenia was described in 22, and anemia with hemorrhages in 20 of the 24 cases. Moore and Keidel called attention to the similarity of the blood pictures in stomatitis, dermatitis, and aplastic anemia resulting from the use of neoarsphenamin." Other cases following the use of the arsenic preparations have also been reported. Foster's patient made a good recovery.

Potts has reported three cases and summarizes them as follows: "I have discovered nothing new in the three cases reported. All the patients were women, and in each case the general health of the patient had been lowered for some months before the onset of the angina. The degree of prostration was out of proportion to the apparent lesion. This was expressly so in case 3. The four cases reported from Omaha in one twelve months' period indicate that the condition may be more common than was before suspected. The differential diagnosis depends on the blood picture. In two of Potts cases examinations of the blood made previous to the onset of the acute condition showed a lowered white cell count and a disproportion of lymphocytes. Several other cases reviewed showed the same condition. A different organism was found in each throat, and there were none in the blood stream. Hence, deductions as to a specific infection cannot be made. A report on the condition of the bone marrow taken from case 3 coincides with the theory that the disease probably is due to some toxin that destroys the granulocytic blood cells and inhibits the blood forming centers of the bone marrow from further production. The treatment given did not have any effect on the course of the disease."

Roberts and Kracke have reported one case. The patient survived the first acute attack, but a subsequent subacute attack proved fatal. They emphasize the following points: "The red marrow of the bones makes erythrocytes, granular leukocytes and platelets. In agranulocytosis, the factory that makes the granulocytes has shut down. The life of this cell in the normal conditions of health is three days or more, and from three to five days by other evidences. Therefore should the red marrow stop manufacturing them for three, four or five days, they would after that time totally disappear from the blood stream. This is apparently what happens. In our patient there is evidence that it did happen. The factory divisions of the marrow that make the erythrocytes and the platelets seem to continue to work at approximately their normal output. There is little if any evidence that the granulocytes are formed normally in the marrow and destroyed in the blood stream. On the contrary, there is much evidence that the myelocytic function of the marrow stops and that the primary pathologic condition is in the bone marrow."

Doctor Windham's use of chromium trioxide and hydrogen peroxide locally is commendable, but I doubt the wisdom of using neoarsphenamin intravenously after reading Foster's article and his review of the literature of similar cases. Small doses may be beneficial, however. The

patients are desperately ill and the attending physician is justified in using any remedy which offers a reasonable chance for improvement.

Dr. J. B. Potts, Omaha, Nebraska: A year ago in Washington, at the meeting of the "Triological Society," I reported three cases. In addition to these there have been three other cases in Omaha, reported by Dr. Sachs, which I have not seen. These three cases were of the fulminating type, and all died in from one to three days after he saw the patients. All had massive destruction of tissue, very low blood count, practically no granulocytes.

The third case of my series, which Dr. Spencer called to your attention, was particularly interesting from the standpoint of possibly throwing some light on the etiology. This was a woman about 45 or 50 years old, apparently in good health, who had been in one of the hospitals three months previously for an attack of asthma, at which time a white-cell count was made which showed from 7,000 to 8,000, with no particular disproportion of granulocyte cells nor manocytes. She had a slight attack of sore throat about one week before she came to the hospital, in which the right tonsil was involved. Three days before we saw her the left tonsil became sore and her husband, who was a physician, brought her to the hospital. There was a slight swelling on this side of the throat, and blood cultures were made not suspecting an agranulocytic condition because there was no ulceration of the throat. I made an incision in the usual place for a peritonsillar abscess, no pus was present, but the black surface showed at the time. I then asked for a white count, which showed 600 white cells,—only one granulocyte was found in the examination of three or four smears. The throat remained much the same for four or five days. Dr. Spencer remarked that these agranulocytic cells have life for only three or four days, but in this case the condition had been present for three or four days before she came in. The blood count dropped daily until by the fifth day she had only 200 white cells. In this patient we found two granulocytes. She died on the sixth day, without any special lesion showing in the throat. The wound I made in the throat stayed as it was on the first day; it did not break down into a sloughing mass. Clinically, we suspected she had developed lesions of the alimentary tract. She did not have any lesions on the surface of the body or mucous membrane. She had 4,500,000 red cells, and 75 per cent. hemoglobin when she came in. The red cells did not decrease and the hemoglobin only slightly because she was not taking a sufficient amount of food.

There was nothing special in the other cases, except that they were under the care of a very careful clinician who was a laboratory worker, Dr. William N. Anderson, who made several careful blood examinations, and at no time did they have a normal blood count.

Dr. F. H. Newton, Dallas, Texas: In view of the great amount of discussion concerning the cause of agranulocytic angina and its status as a disease entity, the proposals of Dr. Windham are indeed interesting and unique, but an analysis of other reports of such cases causes considerable hesitation in agreeing with all of his conclusions.

Ordway and Gorham in their 1930 edition of "Diseases of the Blood" have collected 106 reported cases, making a detailed study of 82 cases which were described sufficiently for analysis. Of the 82 cases, 73 died, the average duration of the disease being four to ten days, the longest ninety days. In three of the cases normal blood counts were found only a few days before the onset of symptoms. In all three cases changes in the blood were noted before the changes in the tissues, which rather points to the fact that the blood disturbance is primary and the tissue changes secondary. Hutcheson of Richmond says that in many instances the mouth and throat lesions have appeared after the disease was well under way.

The role of Bacillus pyocyaneus in the etiology is questionable. This particular organism is frequently found where sloughing tissue is present and is most likely a secondary contamination. The rather widespread variety of organisms reported on smear and blood culture add to the uncertainty of the specificity of any one organism.

A consideration of such diseases as acute poisoning (with benzol, thorium, arsenic, etc.), acute leukemia, infectious mononucleosis, aplastic anemia, and certain cases of severe sepsis, in all of which is present a severe bone-marrow disturbance, and in all of which may frequently be found mouth lesions resembling those of agranulocytic angina, make it easy for one to place agranulocytic angina in this group. Ordway and Gorham state: "It is believed that agranulocytic angina is merely an unusual response of the individual to septic infection, the reflection in the bloodstream of an overwhelming bone-marrow injury, which may depend either upon a marked lessened resistance of the patient or a greatly increased virulence of the invading organism, or upon both factors." The bone-marrow in this particular disease reacts by a marked diminution or absence of the granulocytic cells with little disturbance of the red blood cells or blood platelets.

The difference in the exposure to fresh air between the nose and the tonsillar region is hardly sufficient as an argument for the anaerobic theory of causation. The local treatment outlined should be of considerable value in loosening and clearing up the slough, but one wonders what value it has in arresting the fundamental disturbance. Yet it must be admitted that treatment usually has not been successful. Friedman reports recovery of four cases treated by exposure of the long bones to X-ray. Also several cases of spontaneous recovery with no treatment have been reported. Despite any differences of opinion, however, the fact remains that three out of four cases reported by the essayist recovered. He is certainly to be congratulated.

Dr. Francis Lederer, Chicago: From an observation of approximately fourteen cases, with two recoveries, I hesitate to accept the term angina agranulocytotica, but rather think of the angina as a part of the agranulocytic picture. Our first case occurred in 1922, when we were not aware of this particular type of disease, and we were then more interested in the leukemia. The very fact that this disease is so often confused with Vincent's angina is perhaps a bad thing.

As Dr. Spencer has indicated, it is quite certain that arsphenamin, perhaps not as arsphenamin alone but as a foreign protein, has something to do with precipitating this blood picture.

Dr. Windham has said that this disease is not common in children. Similar to the case of arsenic therapy that Dr. Spencer mentioned a great many cases have been reported in the literature as occurring in children following the use of this remedy.

Dr. Windham also states that toxemia is a secondary manifestation. However, it is not quite clear from a study of these cases or from the literature whether this is true or not. We do feel that angina is a secondary manifestation and that the toxemia is part of the general destruction process.

The essayist also mentioned that this disease begins with an angina. That also is a little out of line with our experience. We were able to observe some of our cases early. Not all cases began with the ulceration the essayist mentioned. Pathognomonic is the peculiar appearance of the tonsil involved. The hemorrhagic appearance is rather a dark red color, demarcated, and perhaps the next day you will notice that area is of a gangrenous nature.

The fact that the doctor's cases had an icterus brings to my mind that when a case of this sort develops an icterus it is usually a bad prognosis. The cases the essayist reports of this angina agranulocytica *have not an absence of polymorphonuclear cells, and it is questionable whether all of these are of a true agranulocytic type.* In agranulocytosis there is a complete absence of polymorphonuclear leucocytes.

Furthermore, we must remember, as Dr. Potts has shown, that these cases are subject to relapse, and perhaps a great many of them are part of a cycle, and if our patients lived long enough they would develop an aplastic anemia. So it is questionable whether agranulocytosis is the primary condition.

The cleansing therapy as recommended by the essayist we think is good, and it may be they were doing something of a rational character, namely, removing toxic material. Perhaps it is a good idea that such therapy be carried out.

One thing I want to add, namely, that the X-ray has proven of benefit in one of our cases applied to the long bones and to the spleen.

Dr. John J. Shea, Memphis, Tenn.: Today, laryngologists see more cases of agranulocytic angina, either because the disease is more frequent or because the laryngologist is more alert. There are two varieties, one that ends fatally and is most frequently seen in middle-aged women, and another that is only a complex. The etiology of the disease is not definitely known. Various observers have isolated the following organisms from lesions of the throat in these cases: *Bacillus pyocyaneus*, hemolytic and non-hemolytic streptococci, staphylococci, and Vincent's bacteria. The toxin that causes the malady is capable of paralyzing the hematopoietic system, especially the bone-marrow, where the myeloblasts are producing granular units. The action of the toxin is the same as that of massive doses of Roentgen rays on the long bones. The initial sore throat is the usual portal of entrance of the infection, but later

other mucous membranes, as the intestines and vagina, present lesions. The throat picture depends on the amount and the location of the lymphoid tissue that it contains, and if the patient has retained her tonsils they will be found to be covered with an exudate. Thereafter, the progress of the disease is that of general infection which ends fatally.

The intravenous administration of arsenic compounds may at times depress the hematopoietic system and present a symptom complex resembling acute agranulocytic angina.

Case 1.—Mrs. D. P., aged 26, who was seen in consultation, had an acute condition in the throat, a fever and a rash previously diagnosed as "measles." During a physical examination her blood was found to give a four-plus reaction to the Wassermann test, and arrangements were made for a course of neoarsphenamin. Three days after the second dose a fever of 104° F. developed, with a macular rash.

On admission to the hospital three days later she still had a slight rash and her blood count was reported as follows: red cells, 3,300,000; hemoglobin, 60 per cent.; white cells, 4,400; polymophonuclear neutrophils, 2.5 per cent.; polymorphonuclear eosinophils, 2 per cent.; small lymphocytes, 81.5 per cent.; large monocytes, 14 per cent. Treatment consisted of one blood transfusion of 300 cc. of whole blood, the intravenous administration of 7½ grains (0.78 Gm.) of sodium thiosulphate every twelve hours for three days and a supportive diet rich in vitamins.

A chart of her blood showed the gradual increase of the granular elements, and the blood count on discharge was: white cells, 9,100; polymorphonuclears, 75 per cent.; small lymphocytes, 25 per cent.; large monocytes, 1 per cent. The course of the blood is shown in Table 1.

TABLE 1.—OBSERVATIONS ON THE BLOOD IN CASE 1.

Date, March, 1930	Hemoglobin, per Cent	Erythrocytes, per C.Mm.	Leukocytes, per C.Mm.	Polymorphonuclear Neutrophils, per Cent	Eosinophils, per Cent	Basophils, per Cent	Small Lymphocytes, per Cent	Large Lymphocytes, per Cent	Large Mononuclears, per Cent	Transitionals, per Cent
11	60	3,300,000	4,900	2.5	2	....	81.5	....	14	....
12	....	4,100,000	4,100	2	1	....	56	3	36	2
13	....	....	4,300	4	....	....	64	2	30	....
14	....	4,100,000	4,650	8	....	....	68	1	23	....
15	....	....	4,550	13	....	....	61	1	24	1
16	....	....	4,550	22	....	1	62	....	15	....
17	....	....	5,000	36	....	....	64	....	10	....
18	....	....	5,200	37	....	....	58	....	5	....
19	....	4,900,000	5,100	49	....	....	42	....	9	....
20	....	....	6,500	52	....	....	42	....	6	....
21	....	....	7,250	60	....	....	36	....	4	....
22	....	....	7,500	62	....	....	32	....	6	....
23	....	....	9,100	74	....	....	25	....	1	....

The following case is one of agranulocytic angina of unknown cause.

Case 2.—W. S. M., a man, aged 60, was admitted to the hospital convalescing from pneumonia. He had a bilateral acute maxillary sinusitis which improved under simple irrigation, and he was ready for discharge when acute angina with a rising temperature developed. A small ulcer was visible on the left tonsil, from which many fusiform bacilli and spirochetes of Vincent were obtained. The blood picture was a revelation: white cells, 1,800; polymorphonuclear neutrophils, 1 per cent.;

small lymphocytes, 97 per cent.; large monocytes, 2 per cent. The granular elements were reduced to 3 per cent., showing that the hematopoietic tissue, the function of which is their production, had been paralyzed. Table 2 demonstrates the course of the observations on the blood.

TABLE 2.—OBSERVATIONS ON THE BLOOD IN CASE 2.

Date, March, 1930	Hemoglobin, per Cent	Erythrocytes, per C.Mm.	Leukocytes, per C.Mm.	Polymorphonuclear Neutrophils, per Cent	Small Lymphocytes, per Cent	Large Lymphocytes, per Cent	Large Monocytes, per Cent	Lymphoblasts, per Cent
22	80	4,000,000	1,800	1	97	....	2	....
23	....	....	2,150	3	96	....	1	....
24	....	....	1,550	2	90	....	4.5	3.5
25	....	3,700,000	1,550	3	82	8	5	2
Transfusion								
27	....	....	3,950	28	60	3	8	1
28	....	4,600,000	4,000	41	52	2	5	....
29	....	....	4,300	48	40	5	7	....
30	....	....	6,300	58	33	....	9	....
31	....	4,500,000	6,900	58	40	....	2	....
April 1	....	....	11,100	72	24	....	4	....
April 2	85	4,500,000	10,500	73	20	....	7	....

DR. AUSTIN A. HAYDEN, Chicago: A point of the first importance that has been touched upon by nearly everyone who has discussed the paper but has not been specifically mentioned by anyone, is the fact, I believe, that a white blood count should be made more often in cases of sore throat. I have seen four of these cases, and all four died. All four had a history of syphilitic infection and a history of arsenic medication to a greater or less extent. One of these showed a diphtheritic membrane with diphtheritic organisms isolated from cultures. Three days before the case terminated, sixty hours after I saw her, she looked as well as I had ever seen her. The variety of bacteria that was isolated from her throat were almost innumerable, but the prominent one was the diphtheria organism.

I think as I look back over that case, of another thing that is not mentioned, and that we did not use, and that is very mild suction in the throat, because one of the most disturbing features of that woman's illness was the fact that she had a large amount of nasopharyngeal secretion, and had great difficulty in relieving herself, on account of the swelling of the glands of the neck and the swelling of the throat itself.

All of the cases I have seen have had white counts of less than 1,000, with almost entire disappearance of the polymorphonuclear cells. The case I reported had a count of 400 with only 2 per cent. the first day. She had a little increase in polymorphonuclears after the first transfusion, a small increase after the second transfusion—perhaps due to the polymorphonuclears contained in the transfused blood. All of these cases had absolutely clean mouths aside from the local lesion on the tonsil. I believe that many of us will avoid a great many unpleasant surprises if we do a white blood count routinely in cases of sore throat.

DR. THOMAS E. CARMODY, Denver: I have used much the same treatment for cleansing the mouth—also peroxide of hydrogen and perborate of soda followed by potassium permanganate, but do not consider it a specific.

I must disagree with the doctor that the condition is local. Having seen a large number of mouth cases in the last twenty-five years, I have seen cases that resemble some of these very closely, but examination from the laboratory standpoint *is not sufficient in my opinion to prove these last three cases agranulocytic angina.* The cases we have seen have not all originated in the throat; in some of them the local lesions have been more in the mouth than in the throat.

I must also disagree with his published article as to the blood supply. I believe it is not so poor around the teeth and tonsils as it is in some other parts of the body, and we do not think that it is any worse than in the nasopharynx.

The use of arsphenamin in these cases, as reported by Foster and others, seems to give us a folliculosis with lack of polymorphs, just the same as the infection.

DR. R. W. WINDHAM, closing: I wish to thank the members who discussed the paper for their explanations that help to clarify the situation. This disease was discovered only in 1922, and since that date it seems to have increased in prevalence day by day. I think success lies in early diagnosis and instituting treatment at an early date. When we see cases late, I do not think any treatment known at the present time will benefit the patient to any extent. Treatment should be instituted rigidly and fearlessly, because we know most of the cases terminate fatally, therefore we have everything to gain and nothing to lose. We have adopted the policy in all cases of giving the parents and relatives a guarded prognosis, remembering to tell them the history of the condition and how we think it might result.

Concerning arsphenamin as a causative factor, we have been using it in the treatment of a great many other conditions, but this condition has only been known since 1922, and if arsphenamin is to blame it looks as though we might have had some of it before that time. My first case had extensive arsphenamin treatment and showed a presyphilitic condition.

After I sent out this questionnaire and got a lot of replies I found a large number of cases of socalled agranulocytic angina that have not been published or appeared in the literature, many men having had one to four cases.

As far as the cases we reported being agranulocytic angina, it was my personal opinion that they were. My idea in presenting this paper was not so much to teach as to try to learn something. The condition is becoming more and more prevalent every day and it should be discussed if we are to solve the problem as to the cause and etiology and probable treatment of these cases.

INTERESTING OBSERVATIONS IN MORE THAN ONE HUNDRED EXTERNAL ETHMOFRONTAL OPERATIONS.

W. Likely Simpson, M. D., and Sam H. Sanders, M. D.,

Memphis.

The object of this paper is to present a number of observations made in a series of over a hundred cases of ethmoid and frontal sinusitis that were operated upon by the external route. The operative technic was ably described by Lynch in 1921, and only a few modifications have been made that do not alter the fundamental principles of the operation. Practically all of these cases have been operated upon under local anesthesia with morphin and scopolamin analgesia, and the interesting points in the latter will also be mentioned.

PREOPERATIVE ANALGESIA.

The preoperative administration of hypodermic injections of morphin and scopolamin in varying doses, depending on the size, age and mental state of the patient, is considered very important. Formerly morphin sulphate, one-fourth grain, and scopolamin hydrobromide, one one-hundred-and-fiftieth grain, were used. Within the last two years we have found that much larger doses can be safely given to individuals between the ages of eighteen and fifty. Patients below the age of eighteen and above fifty must be considered individually, as some can tolerate morphin better than others. The weight of an individual must be considered. We have found that an obese one, of hypothyroid type, cannot tolerate either drug as can the more active and energetic type. The easily excited person is very susceptible to scopolamin but reacts nicely to small doses combined with larger doses of morphin. The patient that fears the operative procedure tolerates both drugs well, probably because the fear is only a temporary state of excitement. In administering one-quarter grain of morphin and one one-hundred-and-fiftieth grain of scopolamin or one H. M. C. tablet number one, at forty-five minute or one hour intervals, three doses can be administered to the average patient, weighing 150

pounds or over without risk, the last dose being given ten minutes before the patient goes to the operating room. Only three cases (two of which were physicians) have shown untoward effects, these being manifested by a form of mania and unwillingness to remain quiet. The sensations of falling and smothering were present. None of these three patients remembered any event that took place in the operating room. One had to be given a general anesthetic, one was restrained by force, the other by persuasion. The average patient became sleepy, unconcerned and often expressed himself as being drunk. There has been practically no complaint of pain during operation, yet the cough reflex is not abolished and the patient can expectorate any blood that may enter the throat. The respiratory rate is diminished, but it can be raised, when desired, by arousing the patient or engaging him in conversation. We have often been cautioned to beware of scopolamin idiosyncrasies, but we have been unable to find a case report of scopolamin having been proven to be the cause of a patient's death. In administering this form of analgesia, we see, or have our nurse see, the patient before the last injection is given, and if necessary the dose of either drug may be increased or decreased according to indications. One patient was given one and one-half grains of morphin and one-seventy-fifth grain of scopolamin before the desired effects were obtained. No ill effects were noticed.

LOCAL ANESTHESIA.

One or two cubic centimeters of novocain is injected into the region of the incision. A larger quantity distorts the parts enough to make one wonder where the incision will be when the solution is absorbed.

Narrow gauze strips saturated with a solution of 4 per cent cocain and equal parts of adrenalin are packed in the wound to prevent bleeding or to anesthetize the tissue when either is desired. We have not seen any toxic effects from cocain when it is used combined with large quantities of adrenalin.

GENERAL ANESTHESIA.

Much more bleeding occurs under general anesthesia. The anesthetist is usually in the way at one time or another during the operation. The patient is unable to cooperate with the operator and many other disadvantages have been encountered in the use of general anesthesia.

TECHNIC.

The entire face is painted with a solution of 3 per cent mercurochrome in a mixture of acetone, 10 per cent, alcohol, 55 per cent, water, 35 per cent. Skin infections have occurred less frequently than when prepared with 3 per cent tincture of iodin or picric acid.

INCISION.

The incision is most easily described with reference to the internal canthus of the eye. The skin is fixed with a finger of the left hand. An incision is made along an imaginary line half the distance between the inner canthus of the eye and the bridge of the nose, beginning about on a level with the inner canthus, extending upward and curving with the orbit well below the brow. The length of the primary incision depends upon the size of the frontal sinus, but it should not extend past the supraorbital notch, as the mucous membrane can often be removed from the frontal sinus without cutting the supraorbital vessels and nerve. In a case with an average sized frontal sinus, the incision is from three-eighths to five-eighths inches in length. Because of the looseness and mobility of the skin in this region, the skin can be retracted and the incision extended through the underlying tissues. A large exposure is obtained where only a small skin incision is made.

HEMOSTASIS.

About four retraction sutures are inserted at this time through the skin and the soft tissue. A piece of gauze wet in the cocain and adrenalin solution is then packed into the wound to stop bleeding. No vessels are tied. We formerly produced hemostasis by ligating these small vessels with catgut, but it has been found that the cocain and adrenalin will accomplish this practically as well, and when a persistent bleeder does occur it can be clamped with a hemostat and thus a dry field is obtained without the use of any suture material. The orbital periosteum is elevated from the ethmo-orbital wall. The anterior ethmoid vessels and nerve are cut at this time to prevent the orbital periosteum from tearing as it practically always does when the orbital structures are retracted before the vessels are cut. Formerly much care was taken to ligate the anterior and posterior ethmoid vessels. An instrument was even devised for this purpose. It seems to us that

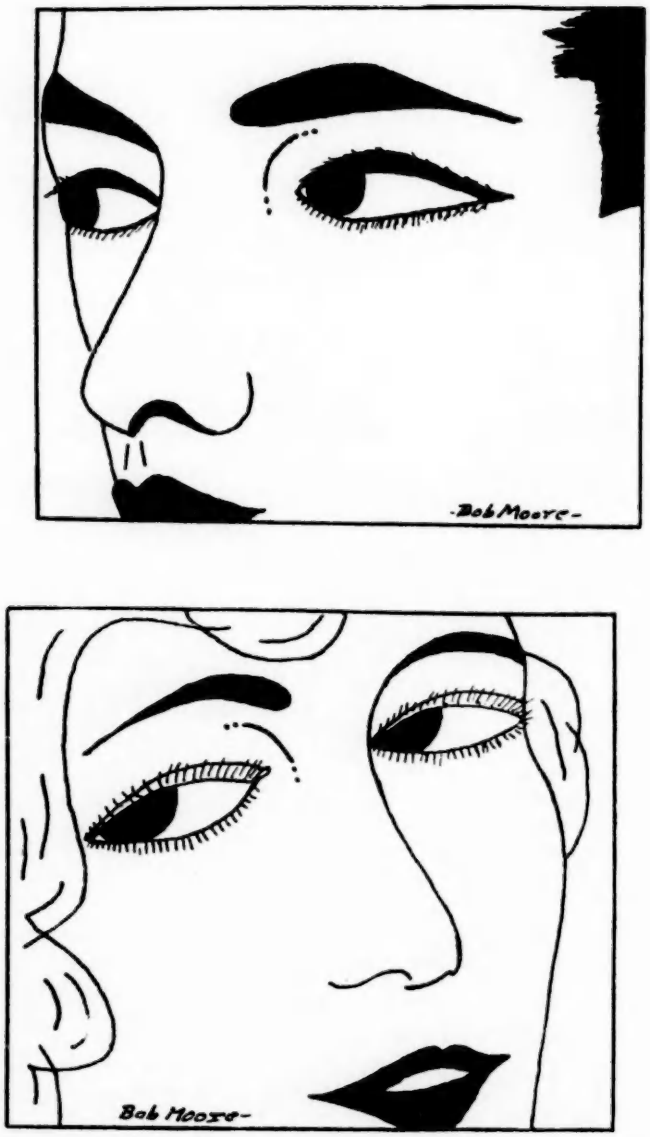


Fig. 1. Schematic drawings to illustrate line of skin incision. Solid line represents the usual line of incision. The extended dotted line represents the enlargement of the incision beneath the skin.

this procedure is seldom necessary. If a small piece of gauze wet in the adrenalin and cocain solution is allowed to remain over these vessels for a few minutes after they have been cut the bleeding will usually stop. This gauze can be left while the operator elevates the periosteum from the frontal floor. If the bleeding persists after this has been done, the vessels can be picked up with a small hemostat and ligated very easily. In some of our more recent cases, and especially those with large ethmoid and frontal sinuses, where more of the ethmo-orbital wall has been preserved and the anterior ethmoid vessels have been disturbed, it has been noted that much bleeding occurs while working in the anterior ethmoid cells. If the anterior ethmoid vessels are cut this area is practically bloodless.

REMOVAL OF FRONTAL MUCOSA.

After opening the frontal sinus and removing the floor an attempt is made to elevate and remove the lining membrane of the cavity. The edge of the membrane is elevated and dry gauze is packed under it. At times the entire membrane can be removed intact. Small mirrors are used to view the angles and corners of the sinus. If any tissue is left it can be removed with curettes. Partial bony septa are removed when possible, and especial attention is given the angles and nooks made by these partitions. It is our impression that the floor of the frontal sinus does not have to be removed at the outer angle if the entire membrane can be removed without it. Only when all the mucous membrane cannot be removed from the outer angle is it necessary to cut the supraorbital vessels and nerve in order to obtain more exposure for removing the floor and any of the remaining mucosa. In fact, we have found all of the larger frontal sinuses very deep and therefore easy to rid of lining. The use of mirrors for the purpose of viewing the recesses, as well as the entire sinus, has been a great aid in removing the small pieces of tissue, which is so necessary to obtain desired results. We feel that the use of these mirrors is an important step in the operative procedure.

ETHMO-ORBITAL WALL.

In the cases with large frontal and ethmoid sinuses as much of the ethmoorbital wall as practical is left intact to prevent the displacement of the orbital contents. Less retraction of the eye has been noticed in the cases where this has been done.

TEARS IN ORBITAL PERIOSTEUM.

The orbital periosteum is often torn but no complications have been known to arise except probably an ecchymosis which is of little importance. In a few cases the orbital periosteum was adherent to the bone. The bone was rough and it was apparent that a previous periostitis had existed. We have obtained specimens of the bone with attached mucous membrane of the ethmo-orbital wall and also the bone and mucous membrane of the floor from the frontal from most of these cases and hope to present later a report of a pathologic study of these specimens.

ETHMOID.

We have found the external approach to the ethmoids a great advantage over the intranasal, for thorough exenteration. One obtains a better view of the operative field, landmarks are definite and the supraorbital cells can be exenterated as well as those small anterior cells around the frontal duct. One or more of these latter cells have been found intact in practically all of the cases that have had intranasal ethmoid operations, even though the operator was considered very competent.

SPHENOID.

No advantage is seen in the external approach to the sphenoid, but the anterior wall can be easily removed on completing the ethmoid exenteration. We have observed less tendency for granulations to form around the sphenoid opening, less discharge in the nose and more rapid recovery in those cases where the sphenoid mucosa was removed. Bleeding has been of little moment.

MIDDLE TURBINATE.

We do not remove the middle turbinate unless it is polypoid or contains infected cells. The attachment of the middle turbinate offers a convenient landmark during the operative procedure. It does not block the drainage and we see no advantage in removing it.

SUBCUTANEOUS SUTURES.

We do not close the periosteum or approximate the subcutaneous structures as formerly, and have even abandoned the suture to reattach the internal oblique muscle, as no benefit seems to have been derived from either procedure. Skin abscesses occur less frequently than when subcutaneous sutures were used.

REMOVAL OF SUTURES.

Since the short skin incision has been used no crosscuts are needed to approximate the corresponding side. A small needle and very fine dermal or horse hair suture material is used. The sutures are removed in twenty-four hours. This does away with pitting or suture marks in the skin and the linear scar is hardly noticeable.

GROSS BONY CHANGE.

In the sinus where gross changes have been noticed in the bone the convalescence is longer, the postoperative discomfort more severe and the nasal discharge persists over a longer period of time.

SEQUELA.

The Edema of the Lids and Orbital Tissue.—Sometimes an edema of the lids and orbital tissue occurs after that following the operation has subsided. This is usually caused by an acute cold or by forcing air into the orbit by sneezing, coughing or vomiting. The swelling usually subsides without any untoward effects. However, in three cases, in one of which the primary operation was done by us, an edema of the eye occurred at intervals over a period of a few years. Symptoms of headache, pain in the eye and the head, nasal discharge, dizziness, diplopia were quite marked. These three patients were reopened, one with very large frontal and ethmoid sinuses. The latter was found thoroughly clean; the opening to the frontal was large but the granulation in the cavity showed some sign of infection. In the other cases there were diseased ethmoid cells present that had not been removed at the primary operation. No edema of the orbital tissues has occurred in any of these cases after the second operation. The last was done about eight months ago.

Diplopia occurs for a short time after the operation, but in this series of cases only one persisted for several months.

COMPLICATIONS.

External Drainage.—In one case the sinus refused to drain satisfactorily intranasally, even though a large Halle probe could be inserted into the frontal sinus. This case was reopened three weeks following primary operation. No obstruction to drainage

was found. The sinus was partially filled with pus and no granulation had begun to form.

Osteomyelitis.—There has not been a case of osteomyelitis in this group, but in a previously reported series there was one patient that developed an osteomyelitis, followed by a brain abscess and death. This patient was operated upon during a subacute exacerbation of a chronic infection, which unfortunately was not pronounced enough to attract attention.

RESULTS.

No deaths have occurred in this group from the operation or from postoperative complications. Recurrent infections are infrequent and those that do occur are mild and of short duration.

Deformity.—In two cases with extremely large frontal and ethmoid sinuses there was a very slight but noticeable retraction of the eye.

Cosmetic Results.—The scar from the incision cannot be seen after a few weeks except on close inspection. There is no bony depression whatsoever. In fact, the majority of the patients state that their friends do not believe they have been operated upon externally.

CONCLUSIONS.

1. Morphin and scopolamin analgesia combined with local anesthesia has its advantages over general anesthesia.
2. A short skin incision gives sufficient exposure for the operation on an average sized frontal sinus.
3. Practically all bleeding can be controlled without the use of ligatures.
4. Removal of the entire lining mucosa of the sinus is required to obtain desired results.
5. A view of the entire sinus cavity is usually necessary to remove the entire lining mucosa, and this can be easily obtained by the use of mirrors.
6. Good results have been obtained where the outer bony angle of the frontal floor has not been removed.
7. Retention of the ethmo-orbital wall prevents a displacement of the orbital contents.
8. The external route offers a safer approach and affords a better view of the ethmoid, especially the supraorbital and those cells around the nasofrontal duct.

9. The middle turbinate is a valuable landmark and does not interfere with operative procedure or good results.

10. Subcutaneous sutures are unnecessary.

11. Poor cosmetic results are no longer considered contraindications for external frontal operations.

DISCUSSION.

Dr. Robert C. Lynch, New Orleans: All of my cases have been operated upon under general anesthesia, but if we stop to discuss that question we will be here a long time.

I cannot, except in one or two instances, remember that I succeeded in accomplishing the results I started for, with an incision as short as that described by Dr. Simpson. I prefer the incision to be long enough to accomplish the thing I am going after. I pack the tissues with novocain and adrenalin in an effort to secure an incision in this locality without bleeding. In my hands, with the incision that I use, I have not been able to accomplish it bloodlessly. The blood vessels must be ligated, and I know of no bloodier incision in nose and throat surgery than one goes through in shaving the narrow side of the nose.

I am in hearty accord with his fourth conclusion, that removal of the entire mucous membrane is absolutely essential to accomplish the desired results. He says a view of the sinus cavity is usually necessary. I say it is *absolutely* necessary in order to remove every vestige of the lining membrane, not only of the frontal sinus but of the ethmoid and sphenoid as well. Every instance where it has been necessary for me to go in the second time to reoperate because of some persistence of symptoms, there has been a retention of some mucous membrane in the outer bony angle. It was for this reason that I laid so much stress on this point in my original article, and I have not changed my mind.

Retention of the ethmo-orbital wall, while it might possibly keep the eye from retracting, it seems to me inhibits the progress of the work. I have only seen the eye really retracted in two instances.

The external route does seem the safer procedure for the ethmo-frontal-sphenoid operative technic, and especially is it necessary in cases where we have orbital projection of the ethmoid cells.

In nearly all instances where I select the radical frontal-ethmoid-sphenoid operation the turbinate has already been removed.

Please remember the opening sentence of my original article: *"After all else has failed, try this." Be sure you do not subject the patient to this operative procedure until every other means of both medicine and surgery has been attempted.*

The replacement of the parts back as nearly to their normal location as possible is a fundamental surgical principle, therefore I still suture the periosteum back as near its original location as possible. This probably accounts for maintaining the orbit in its normal position.

Cosmetic results should be secondary to accomplishing a cure of the disease. I do not now pay so much attention to obtaining a good cosmetic result, but go in and attempt to cure the patient.

Dr. William Mithoefer, Cincinnati, Ohio: I shall limit my remarks to a few pertinent facts concerning ethmofrontal surgery, and show a few slides of anatomic variations.

First, the technic of the operation depends entirely upon the anatomic variations encountered. The complete removal of the ethmoid cells is the most important part of the operation. It is rarely possible to remove all ethmoid cells in intranasal surgery. An isolated frontal sinus infection is never the cause of nasal suppuration. The ethmoid cells are always involved when the frontal sinus is affected. There are many cases where the ethmoid cells alone are involved and require radical exenteration without removal of the mucous membrane of the frontal, inasmuch as this cavity is very often but mildly, if at all, involved. Therefore I do not agree with the essayist when he says it is absolutely imperative always to remove the mucous membrane of the frontal sinus. The removal of all infected ethmoid cells, thereby establishing good drainage, will many times take care of a frontal sinus just the same as a window resection of the antrum ventilates and drains this cavity.

There are other cases where there is no pathology present in the ethmoid or frontal sinus in which the headache is caused by an anatomic variation of the ethmoid cells with closure of the nasofrontal duct. If intranasal measures fail to relieve these patients, an external ethmoid operation is indicated. To obtain healing of pansinusitis complicated with polypi is almost impossible with endonasal measures. The skiagram gives valuable information regarding the extent laterally of the ethmoid cells, and when it shows no ethmoid cells displaced in a lateral position we may expect better results with endonasal surgery. Infection of the laterally displaced ethmoid cells simulates frontal sinus disease to such an extent that a diagnosis cannot be made clinically.

Postoperative retention from closure of the nasofrontal duct occurs more often than one is led to believe. The length of the incision depends on the extent of the lateral and downward displacement of the cells.

Local anesthesia should be the anesthesia of choice unless swelling of the soft parts prevents its use.

Dr. Thomas E. Carmody, Denver: It seems like carrying coals to Newcastle to discuss this paper after it has been presented and Dr. Lynch has discussed it, to say nothing of what Dr. Mithoefer has told us.

We must remember that the frontal sinus is simply an ethmoid which one might say has gone astray and gotten into the frontal bone. As to the results of operation, I wish I could get as good results as these—no deaths in 100 cases. We have had two deaths in the last two years from osteomyelitis which followed, one an operation similar to this, and the other an operation much more extensive. As to cosmetic results, we have followed this procedure for a number of years and find the cosmetic results much better than with any other method. However, as mentioned in the paper and by Dr. Lynch, we must not let the fact that we are not getting good cosmetic results interfere wih a complete operation. Formerly we thought it was not necessary to remove all of the mucous membrane; where it seemed to be normal in some places we allowed it to remain; but some of those cases we have been compelled to reoperate.

Some of them cleared up without complete removal, but today when we operate we try to make complete removal. In some cases we are unable to open all of the ethmoid cells; in some of them we got good results, but in others we had to reoperate. As to the intranasal method, I do not think it is the method of choice with most of us. We feel we can do so much better by the external method.

As to results, we lost one case recently in which this operation was done, but I had an alibi, because the cerebrospinal fluid showed the meningococcus; but my feeling was that possibly we had operated on a case that, if we had waited a day or two, would not have been operated upon.

We can agree with practically all the conclusions of the essayists. There is no question about the use of morphin and scopolamin in some cases, but in cases where there is any great amount of inflammation I think we had better leave them until the inflammation has subsided. Where the patient is especially nervous I feel a general anesthetic in my hands is better. Bleeding is easily controlled. Formerly we thought the middle turbinate should always be removed. Of late we find by infracting it outward and giving good drainage we get results, and unless it is polypoid we leave it. We have found subcutaneous sutures unnecessary. Cosmetic results in my hands have been very good. Many of these cases do not show even on close observation; but of course we "fall down" on some.

Dr. M. M. Cullom, Nashville, Tenn.: The authors say their operation is the same as Lynch's, but I must disagree with them on this point. It is an external operation, but it is not the Lynch operation as I understand it.

First, the question of anesthesia: they have done practically all this series under local, and when they talk about administering a grain and a half of morphin to a patient I get cold chills. And then the use of a 4 per cent. solution of cocain packed into an open wound; I have never had a local anesthetic death, but I have personally known of some very distressing cases where only a small amount of cocain was injected. I have a feeling that local anesthesia is more dangerous than general, and I also feel that 100 cases is not a large enough series from which to draw conclusions as to safety. There is one distinct advantage of local anesthesia—the bleeding is not so profuse. When you have said that you have about covered the case for local anesthesia. After all, the bleeding is very little more than in a simple mastoid operation. But doing an operation of the magnitude of an external operation on the frontal sinuses under local anesthesia is a considerable strain on the surgeon. I think operations under local anesthesia often result in more shock to the patient than we realize. If the operation is done while the patient is unconscious and several hours of sleep follow, much postoperative suffering is avoided.

In regard to the length of their incision, it is not much more than a stab incision. I do not believe I could do an ethmoid and frontal sinus operation through an incision of that size. I am an advocate of an ample incision in any operation. The incision I use is of course depend-

ent upon the size of the frontal sinus, but it extends from the lower bony margin of the orbit along the side of the nose and through the center of the eyebrow as far as necessary. I should say a two-inch incision is as short as I would use.

I am in accord with the authors in regard to hemostasis; also regarding the complete removal of the frontal mucosa. It is necessary to the success of the operation.

They leave as much of the paper plate as possible, another point in which they are in disagreement with Lynch. I remove as much of the paper plate as possible and I have had no case of displacement of the orbital contents.

Like the authors, I have found the external approach to the ethmoids far better for exenteration than the intranasal approach. In regard to the sphenoid, I prefer to open it intranasally, as the authors say, but I must confess that I am afraid to curet the sphenoid; the internal carotids are too close for comfort. If the anterior wall is removed and ample provision made for drainage, I think the sphenoid will clear up, provided all pathologic material is cleared out above.

The authors do not remove the middle turbinate; Lynch removes it completely. I was taught my ethmoid surgery by H. P. Mosher. He told me to remove the middle turbinate, and I do so, and have seen no ill effects.

The authors do not say whether they elevate the periosteum above the line of incision. Lynch does not, and he insists that this lessens the danger of osteomyelitis. The authors do not remove the floor of the sinus out to the external angle; Lynch insists that the removal of the floor shall be thorough and complete. The authors do not remove the entire orbitoethmoid plate; Lynch insists upon its complete removal.

Why am I insisting on these points? Because this article is published with the statement that it is in all essentials the Lynch operation. If recurrences follow an operation performed in this way, the recurrence could not properly be charged to the Lynch operation because the technic has not been followed in detail. If everyone who undertakes this operation changes the technic to suit his own ideas, the operation will soon be unrecognizable and go into disrepute.

Over a period of nine years I have done fifteen external operations on the frontal sinuses, following as closely as I knew how, the technic of Lynch, and the results of the entire fifteen cases have been eminently satisfactory. I know that fifteen cases are not enough to warrant permanent conclusions, but an experience of nine years is worth something, and the Lynch technic has my entire confidence.

DR. ROBERT SONNENSCHEIN, Chicago: I am astonished to hear that local anesthesia is so dangerous. As a member of the original committee of the American Medical Association on local anesthesia, and now as a member of the Permanent Committee to study the toxic effects of local anesthesia, we have learned that cocain is dangerous, but when properly used fatalities and undesirable results are very rare. I trust that Dr. Simpson will at least check up this part of his paper, for while we know we must be extremely cautious in the use of cocain, I would not like

the impression to be broadcast that it is so dangerous that general anesthesia in all nasal work is preferable.

Dr. W. Likely Simpson, closing: I will first consider local anesthesia with reference to its dangers. Cocain 4 per cent. and adrenalin when applied topically is practically nontoxic. Cocain is dangerous if injected into the body. We never inject cocain at any time and take special precautions, such as coloring the solution, not allowing a nurse to fill a syringe, etc., to prevent accidental injection of cocain for novocain.

The preoperative preparation and local anesthesia have been used in most of our sinus cases and not just in this series. In fact, over 3,000 cases have been thus prepared and only those cases described have been unsatisfactory. Each surgeon has his preference and of course there is the case that requires local or general anesthesia, regardless of the preference of the operator, that must be considered.

As stated in our paper, we do not administer large doses of morphin and scopolamin and disregard the patient's reaction to the drug. We use the average size dose at 45 minute intervals, and have someone see the patient between doses.

As to the incision, it should be whatever length one needs for operating, but I notice as time goes on we make our incisions shorter. Formerly when I made an incision through the brow I made a long incision, but at the present time we make a rather short incision, as our cut will show; but the incision through the periosteum is longer than that through the skin, so any part of the field can be reached.

With us, bleeding is one of the mildest features of the whole operation. It really does not enter into our troubles at all, not even when the supraorbital vessels are cut. We simply pack them off for a short time and go on with some other part of the operation. Seldom does it last more than a moment; it is very seldom that we have to tie off any vessels whatever.

The removal of all the mucous membrane is the important step in the whole operation. If the entire mucous membrane is not removed the operation is incomplete. The crux of the operation is to see the entire field, by mirrors or some other method, and the complete removal of the lining mucosa.

I might answer Dr. Mithoefer's discussion on one point, that I cannot cure chronic antral suppuration by making windows.

Continuing with Dr. Lynch's discussion, I think the outer angle is one of the most troublesome parts, but if the entire mucous membrane is removed I do not find that it is necessary to remove the entire floor of the larger sinuses.

The longer I work on the nose the fewer turbinates I remove. I have not removed a turbinate in chronic cases for three or four years unless it was polypoid or contained infected cells. As to sutures, formerly I placed deep catgut sutures in the periosteum and made a special point of drawing the superior oblique into its position, but I use only dermal sutures in the skin and these are removed in 24 hours and no dressing is placed on the wound.

As stated, we also believe that the ethmo-orbital wall should be removed in all cases, except those with very large sinuses where deformity might occur. In these cases the drainage space is much larger with the ethmo-orbital wall in place than in the average case where it is removed.

It seems to me that Dr. Mithoefer's pictures show that many ethmoid cells cannot be removed in an intranasal operation. I am sure that many of us miss the ethmomaxillary cells operating intranasally; these are much better attacked by means of the transantral route.

As to the use of local anesthesia in cases where there is marked swelling, we do not do any radical work where there is acute infection. If there is rupture of the suppuration into the orbit or anything of that type we only do a drainage operation, and then a month or six weeks later if it is necessary we do a complete operation.

I enjoyed Dr. Carmody's discussion very much and would emphasize the fact that cosmetic results are rather important. Every now and then we see a young lady who has had a very long incision, probably one that could just as well have been a half-inch long. We need not worry about the cosmetic results, but I think it is worth while to make them as good as possible.

INSTRUCTIONAL SUBJECTS

INSTRUCTIONAL SUBJECTS

ALBERT H. ANDREWS, M. D., Chicago, Ill.
Diseases of the External Ear.

JOHN F. BARNHILL, M. D., Miami Beach, Fla.
Headaches.

HARRY L. BAUM, M. D., Denver, Colo.
Chronic Purulent Otitis Media. Complications and Treatment.

T. S. BLAKESLEY, M. D., Kansas City, Mo.
Surgical Diathermy.

WELLS P. EAGLETON, M. D., Newark, N. J.
Brain Abscess.

FREDERICK A. FIGI, M. D., Rochester, Minn.
Malignancies of the Oropharyngeal Cavity.

A. C. FURSTENBERG, M. D., Ann Arbor, Mich.
Mediastinal Infections.

CHAS. O. GIESE, M. D., Colorado Springs, Colo.
The Invasion, Clinical Course and Spread of Tuberculosis Throughout the Body.

PERRY G. GOLDSMITH, M. D., Toronto, Canada
Otolaryngology. A Discussion of Various Problems from the Patients' Standpoint and that of General Medicine.

W. E. GROVE, M. D., Milwaukee, Wis.
Examination of the Ear in Head Injury.

SAMUEL IGLAUER, M. D., Cincinnati, Ohio
Peritonsillar, Retropharyngeal, Deep Cervical Abscess and Posttonsillitis Pyemia.

ROBERT C. LYNCH, M. D., New Orleans, La.
Laryngectomy and Laryrgotomy.
Indications for and Technic of the Various Operations.

LYMAN RICHARDS, M. D., Boston, Mass.
Indications for and Methods of Tracheotomy.

ERNEST M. SEYDELL, M. D., Wichita, Kan.
Sinus Thrombosis.

SAMUEL R. SHILLERN, Jr., M. D., Philadelphia, Pa.
Diagnosis and Treatment of Chronic Frontal Sinusitis.

FERRIS SMITH, M. D., Grand Rapids, Mich.
Principles of Plastic Surgery.

ROBERT SONNENSCHEIN, M. D., Chicago, Ill.
Hearing and Tuning Fork Tests.

MR. EDGAR B. BURCHELL, New York, N. Y.
The Anatomy and Its Variations of the Temporal Bone.

T. E. CARMODY, M. D., Denver, Colo.
Plastic Procedures of the Oral Cavity.

GEORGE M. COATES, M. D., Philadelphia, Pa.
Conservative Surgery of the Mastoid.

RALPH A. FENTON, M. D., Portland, Ore.
Ophthalmology and Otology in Aviation.

MORRIS FISHBEIN, M. D., Chicago, Ill.
The Preparation and Writing of Medical Papers.

LEWIS FISHER, M. D., Philadelphia, Pa.
The Diagnosis of Internal Ear Lesions.

CHARLES J. IMPERATORI, M. D., New York, N. Y.
The Esophagus: Anatomy, Physiology and Pathological Lesions.

N. W. INGALLS, M. D., Cleveland, Ohio
The Anatomy of the Internal Ear.

D. C. JARVIS, M. D., Barre, Vt.
The Upper Respiratory Tract as a Guide to Nutritional Disasters.

SAMUEL J. KOPETZKY, M. D., New York, N. Y.
The Problems Present in Meningeal Infections of Otitic Origin.

EDWIN McGINNIS, M. D., Chicago, Ill.
Foreign Protein Therapy in Otolaryngology.

BERNARD J. McMAHON, M. D., St. Louis, Mo.
Histopathology of Mastoiditis.

WM. MITHOEFER, M. D., Cincinnati, Ohio
Treatment of Acute and Chronic Maxillary Sinusitis.

F. J. PRATT, M. D., Minneapolis, Minn.
The Acute and Chronic Ethmoid

JOHN J. SHEA, M. D., Memphis, Tenn.
The Throat and the Hematopoietic System.

ROBERT RIDPATH, M. D., Philadelphia, Pa.
Surgery of the Nasal Septum.

FRANCIS HEED ADLER, M. D., Philadelphia, Pa.
The Modern Viewpoints of Aqueous Formation and Elimination.

THOMAS D. ALLEN, M. D., Chicago, Ill.
The Etiology of Uveitis.

CHAS. A. BAHN, M. D., New Orleans, La.
Business Management of an Office.

WILL OTTO BELL, M. D., Seattle, Wash.
The Modern Ophthalmoscope: Demonstration of Various Types of Modern Ophthalmoscopes, Both Hand and Table Models.

MR. PAUL GOUGELMANN, Chicago, Ill.
The Fitting of a Prothesis.

EMORY HILL, M. D., Richmond, Va.
Ocular Muscle Palsies—Etiology, Diagnosis, Etc.

BEN WITT KEY, M. D., New York, N. Y.
Ocular Complications of the Acute Exanthemata.

JAMES E. LEBENSOHN, M. D., Chicago, Ill.
Recent Advances in Ophthalmic Physiology.

W. H. LUEDDE, M. D., St. Louis, Mo.
Accommodation.

JOHN O. McREYNOLDS, M. D., Dallas, Texas
Comparative Anatomy of the Lens.

C. S. O'BRIEN, M. D., Iowa City, Iowa
Local and Infiltration Anesthesia.

LAWRENCE T. POST, M. D., St. Louis, Mo.
Use of the Thermophore in Ocular Disease.

BERNARD SAMUELS, M. D., New York, N. Y.
Histopathology of the Optic Nerve—Ophthalmic and Histological Pictures.

CHARLES SHEARD, Ph. D., Rochester, Minn.
Discussion on Ophthalmic Lenses, Transposition of Formulæ, Methods of Neutralization, etc.

DORLAND SMITH, M. D., Bridgeport, Conn.
Refraction Without Cycloplegia.

GEORGE F. SUKER, M. D., Chicago, Ill.
Ocular Cerebral Localization.

CHARLES W. TOOKER, M. D., St. Louis, Mo.
Examination of Industrial Eye Cases from the Compensation Standpoint.

H. P. WAGENER, M. D., Rochester, Minn.
Retinitis of Hypertension and Arteriosclerosis.

WEBB W. WEEKS, M. D., New York, N. Y.
Indications for, Technic of, and End Results of Lagrange's Iridosclerotomy.

DAVID W. WELLS, M. D., Boston, Mass.
The Stereoscopic Treatment of Heterophoria.

SYLVESTER JUDD BEACH, M. D., Portland, Maine
Testing of Vision, Including Malingering, Test Charts, Proper Illumination, Notation of Visual Acuity, etc.

ARTHUR J. BEDELL, M. D., Albany, N. Y.
Differential Diagnosis Between Choked Disc and Optic Neuritis.

HOWELL L. BEGLE, M. D., Detroit, Mich.
The Conjunctival Plastic Flap in Perforating Injuries.

CONRAD BERENS, M. D., New York, N. Y.
Use of a Suture in Cataract Operation.

MELVILLE BLACK, M. D., Denver, Colo.
Postoperative Complications and Their Management.

E. J. BRIBACH, M. D., Atchison, Kan.
Degenerative Processes of the Cornea.

JOHN H. DUNNINGTON, M. D., New York, N. Y.
The Clinical Management of Heterophoria.

SANFORD R. GIFFORD, M. D., Chicago, Ill.
Modern Therapy of Corneal Infections, Particularly Ulcers.

ALLEN GREENWOOD, M. D., Boston, Mass.
Tucking Versus Advancement in Squint.

HARVEY J. HOWARD, M. D., St. Louis, Mo.
Foreign Protein Therapy in Ophthalmology.

J. ELLIS JENNINGS, M. D., St. Louis, Mo.
Testing of Color Vision: Various Methods and Their Evaluation.

ARNOLD KNAPP, M.D., New York, N. Y.
Evaluation of the Intracapsular Cataract Operation.

SEARLE B. MARLOW, M. D., Syracuse, N. Y.
Monocular Occlusion Test for Heterophoria.

A. B. REESE, M. D., New York, N. Y.
Demonstration of Glaucoma Lesions (Projection of Specimens).

GEORGIANA DVORAK THEOBALD, M. D., Chicago, Ill.
Lantern Demonstration of Pathological Material.

JOHN M. WHEELER, M. D., New York, N. Y.
Operative Treatment of Detachment of Retina.

MEYER WIENER, M. D., St. Louis, Mo.
Surgery of Lid Deformities.

HARRY W. WOODRUFF, M. D., Joliet, Ill.
Technic of Various Forms of Iridectomy.

SPECIAL LECTURES

PROGRESS IN OPHTHALMOLOGY

DIAGNOSIS AND PATHOLOGY
JOHN N. EVANS, M. D., Brooklyn, N. Y.

MEDICAL AND SURGICAL TREATMENT
CYRUS RUTHERFORD, M. D., Iowa City, Iowa

PROGRESS IN OTOLARYNGOLOGY

DIAGNOSIS AND PATHOLOGY
ARTHUR W. PROETZ, M. D., St. Louis, Mo.

MEDICAL AND SURGICAL TREATMENT
SAMUEL J. KOPETZKY, M. D., New York, N. Y.

PRESENTATION OF INSTRUMENTS AND APPLIANCES

THE BARRAQUER ERISIPHAKE.

Dr. Otis Wolfe, M. D.,

Marshalltown, Iowa.

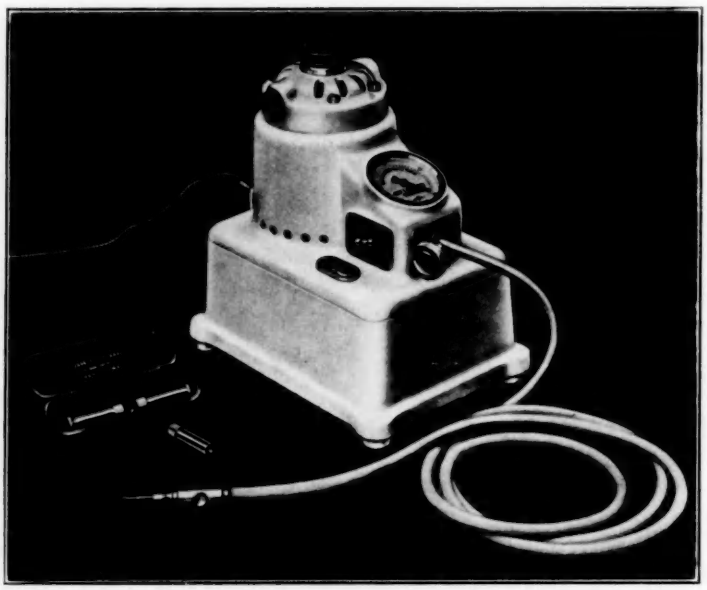
This is the latest model for facoerisis, or the intracapsular extraction of cataract by the method of Dr. Ignacio Barraquer of Barcelona, Spain. He calls it the zonultome and pneumatic forceps.

Dr. Barraquer says this model erisiphake is a great improvement over the previous ones and that he is now fully satisfied with it. It is of much sturdier construction throughout and the electric motor driven vacuum pump is of chromium steel construction, mounted on ball bearings and submerged in oil. Practically no wear is possible and the apparatus is not apt to get out of order in years of use. The vacuum gauge or indicator is constructed entirely of metal and is connected directly with the pump, eliminating all flexible connections. The amount of vacuum can be regulated with the utmost precision.

The cupholder or handpiece containing the valve is made of nickel throughout. The valves are all metallic and highly efficient. There are three cup tips or cannulas of different sizes and shapes. These are made of indium-platinum and will admit of sterilization by heating in a flame. They fit into the handpiece by a conical coupling. The handpieces are beautifully made and are fine examples of expert Catalan workmanship.

Dr. Barraquer and Professor E. Gallemaerts cite some very interesting experiments to prove that the apparatus transmits vibrations to the zonule sufficient to rupture it.

Dr. Barraquer is a prodigious worker and has a first-class mechanical laboratory at his house, where he spends most of his spare time conducting experiments on this and other optical apparatuses.



The Barraquer Erisiphake.

IMPROVED INSTRUMENT FOR ELICITING OCULAR NYSTAGMUS.

PRESENTED BY DR. S. L. SHAPIRO AND DR. S. J. PEARLMAN.

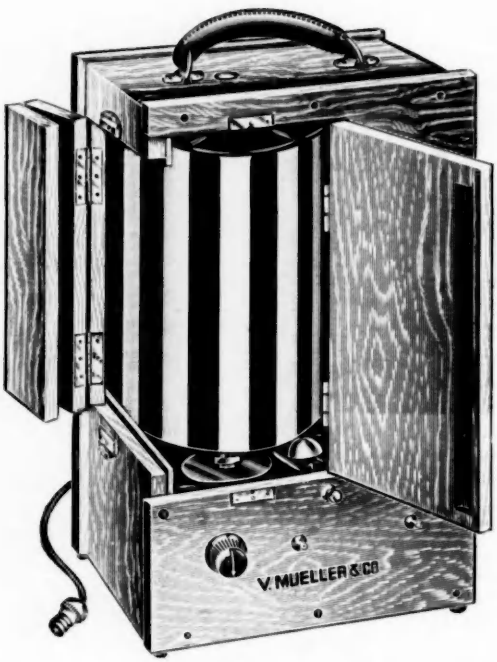
CHICAGO.

The experimental production of ocular nystagmus, which is now recognized as a valuable aid in the diagnosis of certain intracranial lesions, is effected by various methods, all of which are based on the principle of Bárány's original device, namely, that of passing a striped surface before the patient's eyes in a given direction. This instrument employed is a revolving drum, a hoop or an umbrella on which the patient fixes his gaze while the apparatus is rotated before him. While these means are sufficient in most instances to demonstrate the presence or absence of ocular nystagmus they are not adapted for accurate quantitative determinations. It has been demonstrated that the character and speed of the nystagmus produced varies among other factors with the width of the stripes, the interval between them and the rate of speed at which they are passing. To meet these requirements the authors have collaborated in devising an apparatus which is suitable for research as well as clinical purposes. The instrument consists of a metal drum approximately six by fourteen inches, which is rotated by means of a Universal motor and a reducing gear, all contained in an oak case, and so designed that all the working parts of the instrument are easily accessible.

The speed of the revolving drum is regulated by means of a rheostat having more than the average range in that the drum may be revolved from the point where movement is hardly perceptible to approximately 150 revolutions per minute. A combination switch starts and stops the motor and also reverses the direction of the drum, so that it may be rotated either clockwise or counter-clockwise. The instrument may be placed in the horizontal position if it is desired to elicit a vertical nystagmus. If it is desired to know the speed at which the drum is rotating at any given moment, a knob is pulled out, putting in position a bell which rings with each rotation; by means of a stop-watch the time required for ten revolutions is ascertained and the num-

ber of stripes passing per minute in front of the eye can be easily calculated. The drum can be removed and paper jackets with different stripes or pictures employed as needed.

The apparatus, which to our belief, is the only electrical device in use for this purpose, is made by V. Mueller & Co., of Chicago, and our thanks are due their Mr. Wallrich for his assistance.



Improved Instrument for Eliciting Ocular Nystagmus.

THE HUGHES OPHTHALMOTROPE.

PRESENTED BY DR. WENDELL L. HUGHES, NEW YORK.

This ophthalmotrope is the result of a long felt need for a model which will show the normal and pathologic actions of the external ocular muscles, individual or combined, and the effect of operations on any of the muscles.

It is a further development of the model I presented early last year, which was found to be inadequate in some respects. I will briefly mention a few of its features and then show motion pictures of it in action.

In teaching it is useful in demonstrating the relative position of the eyes in the orbits, the anatomy and the effect on the eyeball of active stimulation of the various eye muscles, showing the mechanism of the primary actions of the respective elevators and depressors, and where these primary actions are greatest. The secondary actions are easily explained by showing them on the model. The imitation of normal muscular action is further enhanced by the fact that when the eye is at rest the springs representing the muscles are under slight tension, representing the normal tonicity of the eye muscles; when one "muscle" is activated, that spring is shortened and the opposing ones stretched, imitating the physiologic muscular contraction and relaxation according to Sherrington's law of reciprocal ennervation. The eyes are activated by pulling on strings from the rear.

Any desired paresis or paralysis may be shown by reducing the tension on the corresponding spring by the adjustment of the separate tension strings which are retained by the tension control pegs at the rear.

A rigid member may be substituted for the left lateral rectus muscle to represent the retraction or Duane syndrome. By loosening a small setscrew at the back of the eye a compression spring is brought into play and the eye is allowed to be retracted as well as turn nasally when the medial rectus is activated.

The various operations for weakening or strengthening the actions of the muscles are demonstrable by the attachments pro-

vided, a resection or recession of any desired amount being readily shown. A convergent squint may be shown and then the operations done on the model to correct the deformity. A divergent squint may likewise be shown and corrected.

The use of such a model is particularly for teaching purposes to undergraduates or postgraduate students of ophthalmic myology. It may also be used for demonstrating to patients what can be done at operations on the ocular muscles.

AN INSTRUMENT FOR THE REMOVAL OF ADENOID TISSUE.

PRESENTED BY DR. M. M. CULLOM, NASHVILLE.

This instrument was devised sixteen years ago but has never been shown before any society. It was described in the Journal of the A. M A., March 27, 1915.

To my mind, the best instrument for the removal of adenoid tissue is the curette. It has two drawbacks: The likelihood of losing the growth and the possibility of stripping the mucous membrane off the back wall of the pharynx. Aside from these defects no instrument has been devised which is equal to it.

I had in mind an instrument which would preserve the good qualities of the curette and at the same time overcome the difficulties. The instrument is a sharp curette. In addition it has a flexible blade at the back worked by an arm running through the handle to a thumb ring. There is a movable cage over the fenestrum which permits the adenoid to project through the instrument, enabling one to get down to the very base of the tissue.

The instrument is grasped in the palm of the hand and thrust forward until it touches the vomer. With a downward sweep the sharp blade of the curette is engaged deeply in the growth and then the flexible blade is thrust home and the adenoid is removed with a free sweep just as with a curette. When properly used it removes the growth en masse.

The method of using the instrument is of the utmost importance. Held in the fingers like a pen, as one uses an ordinary adenotome, it becomes just another adenotome. Grasped in the palm of the hand, as illustrated, it removes the growth cleanly and completely.

Instrument for the Removal of Adenoid Tissue.

SUBMUCOUS SEPTUM PUNCH.

PRESENTED BY DR. ROBERT G. REAVES, KNOXVILLE.

This instrument is designed to secure drainage and prevent hematoma following a submucous operation. It makes a little pinhole in the flap—one, two or three—as many as desired. These holes do not close as quickly as one made by a knife, and serve to give proper drainage.

Submucous Septum Punch.

TUNING FORKS.

DR. ROBERT SONNENSCHEIN, CHICAGO.

The latest design of tuning forks presented is made of a new alloy, consisting of approximately 92 per cent aluminum with some copper, manganese and magnesium. These forks do not tarnish and are rustproof.

(For description of other instruments presented, see Minutes.)

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MINUTES.

American Academy of Ophthalmology and Otolaryngology

1930

JOINT SESSION

Tuesday Morning

The thirty-fifth Annual Meeting of the American Academy of Ophthalmology and Otolaryngology was held at the Sherman Hotel, Chicago, October 20th to 24th, 1930.

The Tuesday morning session was called to order at nine-fifteen, the President, Dr. William H. Wilder, of Chicago, presiding.

The President: We are opening now the thirty-fifth Annual Meeting of this American Academy of Ophthalmology and Otolaryngology, one of the largest single societies in existence, one with whose activities you are all well acquainted, and I trust you will find the efforts made by the various committees will prove satisfactory. We have a large hall, but we have amplifiers sufficient, we hope, to enable everyone in the room to hear those speaking from this desk.

The first thing we are to hear this morning is the report of the Chairman of the Committee on Arrangements, Dr. Robert Sonnenschein.

Dr. Robert Sonnenschein: The local committees have done their best to make the arrangements complete, and we trust everything will run smoothly and to your satisfaction. There are a few items that require attention at this time.

In the first place, everyone should be sure to register at the registration booth in the Exhibit Hall.

Second, the ladies will be entertained at a luncheon-bridge at the South Shore Country Club. The wives and daughters of members and guests are invited, and they should register at the booth in the Exhibit Hall, then meet at twelve o'clock sharp, at the east entrance of the Sherman Hotel, to take the busses to the Country Club.

Wednesday evening will occur the annual banquet. Please secure tickets at the earliest possible moment so the management may know how many places to provide.

Tomorrow evening at seven o'clock they will begin to serve, so be there by six-thirty. There will be several short talks, and then a longer address by Prof. Lang, of the University of Chicago, who is one of the wittiest speakers I know of. He will hit off the foibles of the medical men and quacks, and it will be most enjoyable, I can assure you.

The Bausch and Lomb Optical Company are furnishing lanterns and an operator. The Eastman Kodak Company has also given us the use of a projector and an operator. The Zeiss Company are furnishing microscopes.

The Gray Bus Line is making a rate of $1.75 instead of $2.50 for circle motor trips of the city. These may be secured in the west lobby of the hotel.

Please be sure to have your railroad tickets validated by depositing your certificates at the registration desk. If there are as many as 250 the return trip will be one-half fare.

This evening at six-thirty there will be a joint meeting of the Chicago Ophthalmological Society and the Chicago Laryngological and Otological Society. As you know from the program, there will be an address by Dr. Lee W. Dean, of St. Louis, and one by Dr. Walter R. Parker of Detroit. The price is $2.50 per plate. Please inform the registration desk. It is informal and ladies are welcome. We trust to have a large attendance.

Last but not least, I would like to mention a new procedure which will be tried this afternoon. There are two rows of armchairs here in front. This afternoon it is requested that the essayists and discussors, and those who expect to discuss papers, will at the opening of the meeting come forward and occupy these chairs. This will obviate a great deal of loss of time and annoyance during the meeting.

The Second Vice-President, Dr. Frederick A. Kiehle, Portland, Oregon, presided while the President read his Address, entitled "The Standardization of Measurements of Vision and Methods of Recording."

THE PRESIDENT: It is now my very pleasant duty and privilege to introduce the honor guest of this Academy, who has come quite a long distance to be with us. For the information of the members, many of whom know him personally and know his work in ophthalmology, I may say that Prof. de Grósz occupies the distinguished position of Professor in the first clinic of the Hungarian Royal University at Budapest. The Professor comes

of a line of ophthalmologists. His grandfather, over a century ago, established a hospital for the benefit of those who were suffering from eye diseases in one of the towns in Hungary that now belong to the Austrian Empire. His own father also took up special work in ophthalmology and was one of the prominent ophthalmologists of the Austro-Hungarian Empire, and he himself occupies this rather high position in the Royal Hungarian University. So with such antecedents and such special training, which we may say comes in an unbroken line through his forbears, we may expect a man of high attainments. He is distinguished in his field, and I think we may feel honored that he has given us this opportunity and privilege of hearing him.

I have the very great honor and privilege to introduce, as the guest of honor, Dr. Emile de Grósz, of the Royal Hungarian University, Budapest.

Prof. Dr. Emile de Grósz: My warmest thanks for this reception. It is a very great honor, coming from such a little country, to be the guest of the great United States. It is very hard to bring science over to you in a country which has such a high position in the scientific world. I believe you expect me to bring my own experience. I will do my best, and I have only to ask your greatest indulgence.

The following symposium was then presented:

DISEASES OF THE PARANASAL SINUSES AND THEIR RELATION TO DISORDERS OF THE EYE.

Prof. Dr. Emile de Grósz, Budapest, Hungary.

Dr. James A. Babbitt, Philadelphia.

Dr. Sanford R. Gifford, Chicago.

The Tuesday morning session adjourned.

SECTION ON OTOLARYNGOLOGY

Tuesday Afternoon Session

The Tuesday afternoon session was called to order at two-five, the First Vice-President, Dr. Frank R. Spencer, presiding.

Dr. Joseph C. Beck, Chicago, read a paper entitled "Non-Specific Protein Therapy in Otolaryngology." This paper was discussed by Drs. Burt R. Shurly, Detroit; Samuel Levinson, Chicago; Edwin McGinnis, Chicago; Frank J. Novak, Jr., Chicago; and Joseph C. Beck .

Dr. Robert F. Ridpath, Philadelphia, read a paper entitled "Embryology of the Larynx." This paper was discussed by Drs. Ralph A. Fenton, Portland, Oregon; John F. Barnhill, Indianapolis; Thomas E. Carmody, Denver, Colorado; and Robert F. Ridpath.

Dr. French K. Hansel, St. Louis, read a paper entitled "The Diagnosis and Treatment of Allergic Disease of the Nose and Paranasal Sinuses." This paper was discussed by Drs. L. W. Dean, St. Louis; Frederick T. Hill, Waterville, Maine; Frank J. Novak, Jr., Chicago; and French K. Hansel.

Dr. Ralph Almour, New York City, read a paper entitled "The Significance of the Squamous Epithelium in the Cause and Repair of Chronic Middle Ear Disease." This paper was discussed by Drs. Samuel J. Kopetzky, New York City; E. M. Seydell, Wichita, Kansas; S. J. Pearlman, Chicago; Joseph C. Beck, Chicago; Bernard J. McMahon, St. Louis; and Ralph Almour.

Drs. Albert E. and Eugene Bulson, Fort Wayne, Indiana, presented a paper entitled "Chronic Diphtheria Originating in and Limited to the Trachea and Bronchi." This paper was discussed by Drs. Gabriel Tucker, Philadelphia; Millard F. Arbuckle, St. Louis; E. B. Swerdfeger, Denver; and Albert E. Bulson.

The Tuesday afternoon session adjourned.

SECTION ON OPHTHALMOLOGY

Wednesday Afternoon Session

The Wednesday afternoon session convened at two o'clock, with the President, Dr. William H. Wilder, in the Chair.

Drs. Frank E. Burch and H. W. Grant, St. Paul, Minnesota, presented a paper entitled "Some Considerations of Muscle Surgery, with Special Reference to the Tucking Method." This paper was discussed by Drs. W. B. Lancaster, Boston; L. C. Peter, Philadelphia; S. B. Muncaster, Washington, D. C.; Roderick O'Connor, Oakland, California; William T. Davis, Washington, D. C.; and Frank E. Burch.

Dr. J. I. Gouterman, Philadelphia, read a paper entitled "Tay-Sach's Disease: Report of Two Cases." This paper was discussed by Drs. L. C. Peter, Philadelphia; Parker Heath, Detroit; Isadore Goldstein, New York City; and J. I. Gouterman.

Dr. Dorland Smith, Bridgeport, Conn., read a paper entitled "The Estimation of the Total Refractive Error Without a Cycloplegic." This paper was discussed by Drs. S. Judd Beach, Portland, Maine; Edward Jackson, Denver; W. B. Lancaster, Boston; W. H. Crisp, Denver; and Dorland Smith.

Dr. Howard F. Hill, Waterville, Maine, read a paper entitled "Sporothrix Infection of the Eye and Adnexa." This paper was discussed by Dr. A. J. Bedell, Albany, New York.

Dr. Isadore Goldstein, New York City, read a paper entitled "Rosette Formation in the Eyes of Irradiated Human Embryos." This paper was discussed by Drs. E. V. L. Brown, Chicago; William C. Finnoff, Denver.

Dr. Daniel B. Kirby, New York City, read his report as Research Fellow in Ophthalmology, entitled "A Study of Calcium in Relation to Cataract." This paper was discussed by Drs. A. D. Ruedemann, Cleveland; F. A. Davis, Madison, Wisconsin; C. S. O'Brien, Iowa City.

The Wednesday afternoon session adjourned.

SECTION ON OTOLARYNGOLOGY

Thursday Afternoon Session

The Thursday afternoon session convened at two o'clock, the First Vice-President, Dr. Frank R. Spencer, in the Chair.

Exhibition of New Instruments

Drs. S. L. Shapiro and S. J. Pearlman, Chicago, exhibited an improved method of eliciting optic nystagmus.

Dr. M. M. Cuilom, Nashville, Tenn., exhibited an instrument designed for the removal of adenoids. This instrument has a flexible blade which enables the operator to enucleate the adenoids as tonsils are enucleated; also a basket attachment to prevent losing the adenoid in the pharynx.

Dr. Robert G. Reaves, Knoxville, Tenn., exhibited a submucous septum punch.

Dr. Robert Sonnenschein, Chicago, demonstrated the latest design of tuning forks made of a new alloy, consisting of approximately 92 per cent aluminum with some copper, manganese and magnesium. These forks do not tarnish and are rust proof.

Dr. Mark J. Gottleib, New York City, read a paper entitled "Deafness Due to Pancreatic Insufficiency: Symptomatology and

Treatment." This paper was discussed by Drs. Samuel J. Kopetzky, New York City; E. M. Josephson, New York City; and Mark J. Gottleib.

Dr. Frank J. Novak, Jr., Chicago, read a paper entitled "The Besredka Theory of Immunity and Its Possible Application in Otolaryngology." This paper was discussed by Drs. Austin A. Hayden, Chicago; Joseph C. Beck, Chicago; Prof. Lloyd Arnold, University of Illinois; A. W. Morse, Butte, Montana; Ralph A. Fenton, Portland, Oregon; and Frank J. Novak, Jr.

Dr. R. E. Windham, San Angelo, Texas, read a paper entitled "Agranulocytic Angina." This paper was discussed by Drs. Frank R. Spencer, Boulder, Colorado; J. B. Potts, Omaha; F. H. Newton, Dallas, Texas; Francis L. Lederer, Chicago; J. J. Shea, Memphis, Tenn.; Austin A. Hayden, Chicago; Thomas E. Carmody, Denver; and R. E. Wyndham.

Drs. W. Likely Simpson, Memphis, Tenn., and Sam H. Sanders, Detroit, presented a paper entitled "Interesting Observation in More Than One Hundred External Ethmofrontal Operations." This paper was discussed by Drs. R. C. Lynch, New Orleans; W. Mithoefer, Cincinnati; Thomas E. Carmody, Denver; M. M. Cullom, Nashville, Tenn.; Robert Sonnenschein, Chicago; and W. Likely Simpson.

Dr. Richard E. Scammon, Minneapolis, read a paper entitled "Development of Paranasal Sinuses." This paper was discussed by Drs. John J. Shea, Memphis, Tenn.; and Richard E. Scammon.

Dr. Frank R. Spencer: On behalf of the officers of the Academy I want to thank you for your interest, especially in this Section. Dr. Myers tried very hard to arrange an interesting program and we hope you have enjoyed it. The meeting is now adjourned.

SECTION ON OPHTHALMOLOGY

Friday Afternoon Session

The Friday afternoon session convened at two-ten, the President, Dr. William H. Wilder, in the Chair.

Exhibition of New Instruments

Dr. Wendell L. Hughes, New York City, gave a moving picture demonstration of the Hughes ophthalmoscope.

Dr. A. S. Green, San Francisco, presented an optical trephine which is intended to overcome some of the objections to the von Heppel trephine; it is a lighter instrument and much more easily handled; there are three blades of different sizes which are interchangeable. He also presented a cataract mask made of aluminum.

Dr. F. Park Lewis, Buffalo, demonstrated a simple method of application of ardium treatment for painful, noninflammatory conditions of the eye.

Dr. Otis R. Wolfe, Marshalltown, Iowa, presented the new Barraquer pump. It is of much heavier construction than the original and the vibration is improved. As a gauge it accurately controls the amount of vacuum. There are three plates to be used in connection with this pump, of three different sizes and shapes, the objection to them being that the edges are sharp instead of round as in the Green instrument.

Dr. George F. Suker presented a pair of forceps on the order of chalazion forceps, only much larger, for the purpose of making a lid flap for transplant into the conjunctival sac and the eyeball. By screwing it down tight hemorrhage is stopped and the lid held tight while the mucous membrane is dissected. Also a cataract knife, to be used especially in operations for glaucoma.

Dr. Ramon Castroviejo, Chicago, exhibited Dr. Arruga's speculums for the eyelids, one for the right eye and one for the left.

The following symposium was presented:

Symposium on Trachoma

Prof. Dr. Emile de Grósz, Budapest, Hungary.

Dr. J. A. Stucky, Lexington, Kentucky.

Dr. Peter Olitsky, New York City (by invitation).

Dr. Charles Weiss, St. Louis (by invitation).

The above symposium was discussed by Drs. Paul D. Mossman, U. S. Public Health Service, Albuquerque, New Mexico; William C. Finnoff, Denver.

Dr. Allen Greenwood, Boston, read a paper entitled "Lattice Keratitis: Studies of Four Cases Observed in One Family." This paper was discussed by Dr. Richard Gamble, Chicago.

Dr. Otis R. Wolfe, Marshalltown, Iowa, read a paper entitled "The Barraquer Cataract Operation." This paper was discussed

by Drs. Harry Woodruff, Joliet, Illinois; W. B. Lancaster, Boston; S. G. Higgins, Milwaukee; A. S. Green, San Francisco; W. A. Fisher, Chicago; O. B. Nugent, Chicago; Ramon Castroviejo, Chicago; and Otis R. Wolfe.

Dr. Ralph I. Lloyd, New York City, read a paper entitled "Herpes and Allied Conditions." This paper was discussed by Drs. S. B. Marlowe, Syracuse, N. Y.; J. B. Corser, Scranton, Pennsylvania; E. E. Blaauw, Buffalo.

THE PRESIDENT: This closes our program, and while I realize the importance to you of getting away, I want to say that we have a very respectable contingent in size, and certainly a very large one in quality, left.

In closing, the only thing left for me to do is to thank the members of this Section for the evident appreciation they have had of the program we have prepared for them. I want particularly to thank the Secretary and those who have given such excellent work toward the arrangement of the program, and I wish also to express my personal thanks to Dr. Kiehle, the Vice-President, who has assisted me in conducting the program. The meeting stands adjourned.

THURSDAY EVENING SESSION

At five-thirty, Thursday evening, an informal dinner was given, followed by a discussion of the question of forming a Teachers' Section in the Academy for the purpose of providing an open forum for the discussion of questions of especial interest to those members who teach in the various medical schools of this country and Canada.

A formal paper on "The Qualifications of the Medical Specialist" was read by Dr. Harold Rypins, Secretary of the New York State Board of Medical Examiners.

This was followed by informal discussion by Drs. S. L. Kopetzky, Chairman; Edward Jackson, Denver; Allen Greenwood, Boston; Harvey J. Howard, St. Louis; John F. Barnhill, Indianapolis; Harris P. Mosher, Boston; Joseph C. Beck, Chicago; Ralph A. Fenton, Portland, Oregon; Harry S. Gradle, Chicago; Perry G. Goldsmith, Toronto; William H. Wilder, Chicago.

Upon motion, it was decided to present the following resolution to the Council and ask for its favorable action:

WHEREAS, those of our members engaged in teaching the specialties of otolaryngology and ophthalmology have neither a forum for the exchange of ideas nor the means to supervise and when necessary standardize the courses they give, and as a group are inarticulate; and

WHEREAS, it has seemed to us that the formation of a Teachers' Section would supply a need felt by many; therefore,

BE IT RESOLVED, that the Council be instructed to appoint the necessary officers or committeemen to form such a Section, the rules and regulations for the administration of such a Section to be adopted and approved by the Council; and

BE IT FURTHER RESOLVED, that the proper authorities shall enact the necessary measures to provide the finances needed in the formation and functioning of such Section.

ARTHUR J. BEDELL
ARTHUR W. PROETZ
L. W. DEAN
J. A. STUCKY
SAMUEL J. KOPETZKY
RALPH A. FENTON
FRANK E. BURCH
T. E. CARMODY
G. M. COATES

The meeting adjourned.

BUSINESS MEETING

The Business Meeting was held Thursday at nine-thirty, in the Bal Tabarin, the President, Dr. William H. Wilder, presiding.

On motion, the reading of the minutes of the previous meeting was dispensed with.

The following reports of officers and committees were submitted:

REPORT OF SECRETARY

1930

The Academy membership on October 1, 1930, was as follows:

Reported at the Atlantic City convention	1,585
Elected to membership at Atlantic City	126
Total at the beginning of this year	1,711

During this year:

Dropped for non-payment of dues	14	
Deaths	27	
Resignations	3	44
Membership October 1, 1930		1,667

Membership divided as follows:

Honorary Members	12	
Life Members	12	
Carried by order of Council	5	
In good standing	1,580	
One year delinquent	34	
Two years delinquent	24	1,667
Applicants pending		186
Total carried on books		1,853

During the past year the following deaths have occurred:

Dr. R. D. Alway, Aberdeen, S. D.
Dr. Wm. F. Beggs, Newark, N. J.
Dr. D. L. Bettison, Dallas, Texas
Dr. Jesse F. Boone, Chicago
Dr. O. C. Breitenbach, Waukegan, Ill.
Dr. Edmund T. Brown, Burlington, Vt.
Dr. D. H. Coover, Denver, Colo.
Dr. W. L. Dayton, Lincoln, Neb.
Dr. Jacob G. Dorsey, Wichita, Kan.
Dr. Wolf Freudenthal, New York
Dr. Harold Gifford, Omaha
Dr. F. C. Howard, St. Louis
Dr. J. H. James, Mankato, Minn.
Dr. James W. May, Kansas City, Kan.
Dr. John F. Murdoch, Pittsburgh
Dr. Walter Eugene Murphy, Cincinnati
Dr. James A. Patterson, Colorado Springs, Colo.
Dr. James M. Patton, Omaha
Dr. William J. Rideout, Freeport, Ill.
Dr. Ross H. Skillern, Philadelphia
Dr. S. McCuen Smith, Philadelphia

Dr. Louise V. Stegman, Battle Creek, Mich.
Dr. Frederic M. Strouse, Philadelphia
Dr. Derrick T. Vail, Cincinnati
Dr. Lee M. Willard, Wausau, Wis.
Dr. J. M. Woodson, Temple, Texas

The following members resigned:

Dr. L. E. Barnes, Chicago
Dr. Wendell Phillips, New York
Dr. John J. Rainey, Troy, N. Y.

The following members have been dropped for non-payment of dues:

Dr. A. C. Bartholomew, Fort Wayne, Indiana
Dr. Wm. L. Kenney, St. Joseph, Mo.
Dr. G. E. Seaman, Milwaukee, Wis.
Dr. V. E. Van Kirk, Pittsburgh

At the Atlantic City convention the fiscal year was advanced from August first to January first. This change has made it necessary to extend the financing this year over a period of eighteen months, as of August 1, 1929, to January 1, 1931. Because of this change the books will not be audited until after January first, and the report will be presented at the 1931 convention.

From correspondence received in the secretaries' offices it is evident that many members are unaware of the methods followed in the construction of the convention programs—scientific and instructional.

At each convention, and in the announcements, members are requested to submit not only subjects that they themselves are desirous of presenting, but also are asked to report on suitable research work accomplished by others. This information should be forwarded to the respective Section secretary prior to January first, for at that time the newly elected officials assume office, and very shortly afterward the Program Committee is called together. This committee consists of the President, President-elect, First Vice-President, Treasurer, the five Secretaries, and the General Chairman of the local committee for the forthcoming convention.

To this committee are submitted all suggestions contained in the secretaries' files, and each item is carefully discussed. The

general outline is constructed and the papers accepted are those which tend to produce a program of broad, scientific merit. Usually about half the places are filled through invitation; the balance, with the exception of two or four placements, are filled from voluntary offerings. The entire committee functions as a unit, thus eliminating the danger of personal prejudices, unfairness and the many other factors so often encountered where a single individual is vested with authority.

It has seemed to the officers of the Academy (those elected to guide the society's course) that the system adopted serves better to maintain a high type of program as well as to bring to the membership at large, during the annual meeting, the best that is available of the scientific progress of today.

An organization as this Academy, with a membership of nearly 2,000, having activities so greatly diversified, involves an immense amount of detail work, which is handled in the offices of the various secretaries. Since this work is an addition to the professional duties of each secretary, being as it is a labor of love, it is reasonable to expect that errors will creep in. The officers earnestly request members to advise of mistakes detected.

We make one further plea: kindly assist those conducting this work by answering promptly all correspondence.

Respectfully submitted,

WILLIAM P. WHERRY,
Executive Secretary.

Upon motion, duly seconded, this report was accepted and filed.

REPORT OF SENIOR MEMBER OF COUNCIL

DR. HARRIS P. MOSHER: The following is a summary of the action of the Council:

The Council recommended that the Presessional Volume for 1931 be discontinued, and that a liberal abstract of the papers be substituted in the program. It was also decided to ask the members of the Academy, at the 1931 session, to express themselves as to which method they like the better.

The Council approved the changes in the Constitution and By-Laws as given in this year's program. These changes were proposed last year and, according to the By-Laws, come up for action at this time.

The Finance Committee reported favorably on the proposed changes in conducting the business affairs of the Academy. The Council approved this report.

The Finance Committee authorized and the Council approved the payment of $1200.00 to the Army Medical Museum for services rendered the Committee on Pathology.

The Research Committee recommended and the Council approved the award of Research Fellowships as follows: Dr. D. B. Kirby, for ophthalmology, $2000.00; Dr. W. J. McNally, for otolaryngology, $2000.00.

The following Committee Reports have been filed with the Council:

AMERICAN BOARD OF OTOLARYNGOLOGY

October 23, 1930

To the Council of
The American Academy of Ophthalmology and Otolaryngology,
Chicago, Illinois.

Gentlemen:

As members of the American Board of Otolaryngology we wish to submit the following report:

Total number of certificates granted by the Board since its organization, 1,626. Of these 25 are now deceased, leaving 1,601 practicing physicians who now hold this certificate.

As the number passed at the Philadelphia examination was included in last year's report, we can only report on the examination held in Detroit preceding the American Medical Association. At that examination 78 were examined, of which 58 were certificated; 11 failed; 2 were conditioned; 5 passed a junior or preliminary examination, and 2 were held pending completing of their files or cases.

The examination held preceding this meeting is not included in this report.

Respectfully submitted,

T. E. CARMODY
W. P. WHERRY

Committee to Investigate the Treatment of Incipient Cataract

Your committee wishes to report progress. We wish particularly to call the attention of the members to the investigative work of one of the committee members, Dr. Daniel B. Kirby. His paper on the study of calcium in relation to cataract has been presented to the Academy and his results are evidence that the administration of calcium and parathormone do not cause absorption of opacities or improve vision in cases of incipient cataract.

Several members of the committee have been investigating the question along clinical lines and individually are of the opinion that enough is being accomplished in the non-surgical treatment of cortical cataract to encourage the continuance of their investigations.

The committee as a whole wishes to encourage the efforts that scientific ophthalmologists may make toward solving this vexing problem.

We wish to call to the attention of members that on pages 245 to 248, Vol. 5, of the new Kurze Handbuch der Ophthalmologie, edited by Shick and Bruckner, there is a splendid account of the medicinal treatment of senile cataract which summarizes the impressions of most ophthalmologists on this subject.

The committee wishes to be continued.

Allen Greenwood
Hans Barkan
F. W. Dean
Harry S. Gradle
Daniel B. Kirby

Commitee on Hard of Hearing

The standing Committee on the Hard of Hearing reports notable progress in the field of its activities during the past year.

There has been a significant increase in national interest in the prevention of deafness, especially among our school population, largely due to the efforts of members of this and similar committees.

Great progress has also been made in the amelioration of the condition of the deafened through the establishment of many new local organizations for the Hard of Hearing. In Chicago, and in many other cities, largely through the influence of one of our

members, many theatres showing sound pictures have installed individual headphones with high amplification for their hard of hearing patrons. Also many churches are similarly meeting the needs of this large group. Telephone companies throughout the country have, during the past year, made available additional equipment for amplifying the sounds of the ordinary telephone receiver so that many persons long unable to telephone are again able to communicate comfortably over the telephone.

Your committee, having long interpreted its function to consist largely in aiding the movement for the prevention of deafness and the amelioration of its consequences, deems it wise to change its name to the Committee on the Prevention and Amelioration of Deafness. It requests the permission of Council to so change its official name and to continue its activities.

Under authority voted at a previous meeting of the Academy, with the approval of the President, the committee has augmented its membership by the appointment of the following new members: Dr. Austin A. Hayden, Chicago, to succeed the late Dr. Charles W. Richardson; Dr. John F. Barnhill, and Dr. E. H. Cary.

Respectfully submitted,

HORACE NEWHART, Chairman.

COMMITTEE ON OPHTHALMIC AND OTOLARYNGIC PATHOLOGY

There has been no major change in the work of this Committee since the report of 1929. Material is still coming in to the Army Medical Museum at about the same rate as during the past few years, and is being worked up and returned to the donors as previously.

The study sets are in demand, particularly in the smaller towns. It is still too soon to judge of their absolute importance, but letters of praise from those who have used them are being received by this Committee. The sets are being used by the Academy this year for the first time in the Courses of Instruction and are playing an important role in the teaching of ophthalmic pathology. It is hoped that they will be called for to a greater extent next year by applicants who are presenting themselves for the Board Examinations.

Thus the Committee has nothing specific to report except general progress and the hope that more Fellows will avail them-

selves of the facilities offered by this Section of the Academy and the Army Medical Museum.

HARRY S. GRADLE, Chairman.

The following is the report from the Army Medical Museum:

ARMY MEDICAL MUSEUM

October 20, 1930

	Eye Specimens Received and Reported	Eye Specimens Diagnosed When Received	Other Items Illustrations, Models, etc.	TOTAL
For 7 yrs. ending December 31, 1927	800	583	1,124	2,484
For year ending December 31, 1928	177	46	0	223
For year ending December 31, 1929	305	21	0	326
January 1, 1930, to July 1, 1930	193	1	0	194
TOTAL	1,475	651	1,124	3,227

The collection of eye sets has been sent out to 17 individuals during the year. This collection is growing rapidly in importance and the approval of this Academy of the project now under the National Research Council is before you for action.

GEO. R. CALLENDAR,
Major, Medical Corps, U. S. A.,
Registrar.

[An outline of the scope of this registry will be found on page 530.]

REPORT OF AMERICAN COMMITTEE ON OPTICS AND VISUAL PHYSIOLOGY

The President's Address calls attention to a subject that has been under consideration of this committee during the past year. The committee has secured promised co-operation of the Department of Psychology of the University of Michigan, and the United States Bureau of Standards, for the study and standard-

izing of tests for the acuity of vision. The need for and importance of such assistance, and of the general support of ophthalmologists, is evident from the history and present use of visual tests.

It is 250 years since attention was called to the angle of about one minute, that must separate the direction of two stars, or similar objects, to make them appear as separate. It was about 70 years ago that Donders suggested this angle as the basis for more exact visual tests; and Snellen, choosing block capital letters, inscribed each letter in a square subtending the angle of 5 minutes on each side, at the greatest distance at which the letters could be recognized by eyes with good vision.

Three years later Snellen found that some of the letters he had chosen did not conform to visibility, at the distance for which they were numbered—some of the 20-feet letters were visible at 30 feet. He made a re-selection of letters and suggested other figures of like size, including the incomplete square. Later he modified that square by introducing a middle arm, making it the figure now widely known as the "Snellen E."

Since that time capital block letters, modified block letters, Gothic letters and some others have been arranged on test cards and called "Snellen letters," and widely used for testing visual acuity. These cards have varied in the size, shape and visibility of the letters. For some cards the size has been accurately calculated to subtend the five-minute angle at the distance for which the letters were numbered. For other cards the sizes have been inaccurately copied from cards previously published. Cards from most varied sources, with wide variations of accuracy, have thus come into general use; and the results of tests made with all of them have been recorded in the fractional form suggested by Snellen in feet, or in meters. Thus the literature of ophthalmology, the histories of cases, the records of industrial commissions and the courts, have been strewn with fractions, either vulgar or decimal, supposed to designate the distance the tests were recognized, as compared with the distance they should have been recognizable with good vision.

The convenience and value of letters for purposes of testing vision are obvious. But the different letters are recognized by their differences of shape; and these differences of shape make them unequal as to the distances at which different letters can be

recognized. With Gothic letters that have been used for such tests, the differences have amounted to 50 per cent. With carefully constructed block letters, of the same size, the differences amount to 30 per cent. Such variations are intolerable, in a standard by which visual acuity is to be judged: in schools for admission to branches of public service, or in the trial of claims for industrial damage and disability. There is great and immediate need for accurate, reliable, authoritative visual tests, founded on careful, scientific research.

The committee recommends: That the American Academy of Ophthalmology and Oto-Laryngology appropriate from its funds devoted to research the amount of $1,500, or so much thereof as may be needed, to assist in meeting the expenses of such research.

EDWARD JACKSON, Chairman.

COMMITTEE ON STANDARDIZATION OF TUNING FORKS AND HEARING TESTS

Your Committee on Standardization of Tuning Forks and Hearing Tests, which has been in existence since 1922, presents this report:

One of the most important subjects which was considered was that of obtaining forks made of a metal which is rust-proof and which at the same time has the elasticity and other desirable qualities of steel. Two years ago we reported to you forks made of an alloy (consisting of 95.6 per cent magnesium, 0.4 per cent manganese, and 4 per cent aluminum), by Mr. B. E. Eisenhour of the Riverbank Laboratories, Geneva, Illinois. This series of forks extended from C-2 (16 double vibrations) to c-5 (4096 double vibrations). These forks served very well, both for bone and air conduction, with the exception of the c-4 and c-5 forks, which vibrated only a short time as compared with steel forks of the same pitch. The magnesium alloy forks are very light in weight as compared with steel and do not rust. While no peeling occurred, the forks change color due to oxidation.

In order to overcome the short period of vibration of the highest pitched forks, and also to prevent oxidation, further experiments were made by Mr. Eisenhour with a new alloy consisting of approximately 92 per cent aluminum with some copper, manganese and magnesium. This alloy showed many excellent qualities, not the least being its specific gravity of 2.8, as compared

with that of steel, which is 7.7 to 7.9. In other words, it is only about one-third as heavy as steel. In the second place, the forks made of this metal do not tarnish, but retain the silver-like color of aluminum. In the third place, the c-4 and c-5 forks made of this alloy have a very considerable period of vibration, comparing very favorably with a steel fork of the same pitch, even though their vibration period is not quite so long.

These forks, therefore, have the very desirable qualities of lightness of weight, absence of rust or tarnish, long period of vibration for air and bone conduction, elasticity, and, last but not least, a lower cost than the best imported German forks.

Mr. Eisenhour tested the alloy forks for stability of frequency by vibrating them electrically. When thus exciting the c-1 (256 d. v.) fork 500 hours, or 460,000,000 vibrations, and checking the frequency by a method that could detect changes of one vibration in 250,000, no change in the frequency of the fork under test could be noted. We believe that this series of forks will answer all requirements and trust that experience with them will confirm this view.

Owing to the fact that even the very best c-5 forks do not vibrate by air as long as desired, Professor Karl L. Schaefer and Dr. Frank Wethlo of Berlin have devised a metal rod with the pitch of c-5, which is said to have a long period of vibration. We will investigate this matter and will endeavor to report more fully upon it a year hence.

The committee recommends the adoption of this report and the continuance of the committee for further work along the lines of standardization of tuning forks and hearing tests.

Respectfully submitted,

ROBERT SONNENSCHEIN, Chairman
GEORGE W. MACKENZIE
L. W. DEAN
E. G. GILL

COMMITTEE ON EXTRAMURAL GRADUATE INSTRUCTION

This committee was appointed for the purpose of investigating and reporting upon ways and means of promoting extramural graduate instruction in our specialties in addition to that carried on in established institutions.

The committee recognizes that there are three necessary fields of graduate instruction, namely: (1) for the recent graduate who wishes to qualify as a specialist; (2) for the general practitioner who wishes to specialize; and (3) for the practicing physician who is recognized as a specialist in eye, ear, nose and throat diseases, but who desires further graduate instruction.

The requirements of the first two groups can be met only by established institutions for graduate instruction. However, judging from the avidity with which the courses of graduate instruction given by the Academy have been attended for the past ten years, it would seem that there is a nation-wide desire for similar courses which will be more readily available to practitioners throughout the country. These courses must be limited to members of the third group.

The present courses of instruction fostered by the Academy, the Colorado Congress, and others, are insufficient to meet the demand. The committee believes, therefore, that the time is now ripe for the establishment of extramural courses of graduate instruction in various parts of the country under the auspices of organized medical societies and medical schools, in order that a greater number of ophthalmologists and otolaryngologists may have the advantage of such courses with less expenditure of time on the part of each individual.

From the experience gained in the past ten years with the Section on Instruction of the Academy, the committee is certain that the Academy can be of material aid in promoting such extramural courses by: (1) the regulation of the number of hours involved; (2) the supervision of the curriculum; and (3) selection of the instructors.

The committee therefore makes the following recommendations:

1. That the Academy foster extramural graduate instruction.

2. That this function of the Academy be delegated to the Section on Instruction with power to appoint subcommittees.

Respectfully submitted,

W. V. Mullin, Chairman
Harry S. Gradle
Wm. L. Benedict

The following members have been elected by Council and resignations accepted:

Life Members:

Dr. H. B. Young.....Burlington, Iowa
Dr. Otto Stein.....Chicago
Dr. Grant Selfridge.....San Francisco
Dr. J. H. Allen.....Denver

Honorary Member:

Prof. Dr. Emile de Grósz

Resignations Accepted:

Dr. F. N. Irvine.....New York
Dr. J. R. Scott.....Ottawa, Kan.
Dr. R. D. Gibson.....Youngstown, Ohio

Moved by Dr. Ralph A. Fenton that the report of Council be accepted. Motion seconded by Dr. Robert Ridpath and carried.

Necrology Report

The Academy has lost the following members by death during the past year:

Dr. Robert D. Alway, Aberdeen, South Dakota.
Dr. William Franklin Beggs, Newark, New Jersey.
Dr. David Lemual Bettison, Dallas, Texas.
Dr. J. F. Boone, Chicago, Illinois.
Dr. Oscar Carl Breitenbach, Waukegan, Illinois.
Dr. Edmund Towle Brown, Burlington, Vermont.
Dr. David Hummel Coover, Denver, Colorado.
Dr. William Linsley Dayton, Lincoln, Nebraska.
Dr. Jacob Gray Dorsey, Wichita, Kansas.
Dr. Wolff Freudenthal, New York, New York.
Dr. Harold Gifford, Omaha, Nebraska.
Dr. Francis C. Howard, St. Louis, Missouri.
Dr. John H. James, Mankato, Minnesota.
Dr. James W. May, Kansas City, Kansas.
Dr. John F. Murdoch, Pittsburgh, Pennsylvania.
Dr. Walter Eugene Murphy, Cincinnati, Ohio.
Dr. James A. Patterson, Colorado Springs, Colorado.
Dr. James M. Patton, Omaha, Nebraska.
Dr. William Jacob Rideout, Freeport, Illinois.
Dr. Ross Hall Skillern, Philadelphia, Pennsylvania.
Dr. S. MacCuen Smith, Philadelphia, Pennsylvania.
Dr. Louise V. Stegman, Battle Creek, Michigan.
Dr. Frederick M. Strouse, Philadelphia, Pennsylvania.
Dr. Derrick T. Vail, Cincinnati, Ohio.
Dr. Lee M. Willard, Wausau, Wisconsin.
Dr. J. M. Woodson, Temple, Texas.

Respectfully submitted,

H. W. Lyman,
Necrologist.

Dr. Robert Douglas Alway

Dr. Robert Douglas Alway, Aberdeen, South Dakota, died December 21, 1929, at the age of 61 years.

He was one of the Faculty of the University of Toronto Medical School, Toronto, Ontario, Canada, in 1893; a member of the House of Delegates of the American Medical Association, 1909-10; Past President and Secretary of the South Dakota State Medical Association; Past President of the State Board of Health and Medical Examiners; a member of the American Academy of Ophthalmology and Otolaryngology, and of the American College of Surgeons. He was also on the staff of St. Luke's Hospital.

Dr. William Franklin Beggs

Dr. William Franklin Beggs was born in Petaluma, California, on December 30, 1865, and died in Glen Ridge, New Jersey, September 24, 1929. He was the son of Eugene and Catharine Van Winkle Beggs, of Paterson, New Jersey.

Dr. Beggs was educated in the public schools of Paterson, New Jersey, after which he entered Hahnemann College of Medicine in Philadelphia, where he received his degree in 1893. After graduating he went abroad and studied at both the London and Edinburgh universities.

In 1894 he started the practice of general medicine in Roseville, New Jersey, where he remained ten years. In 1898 he graduated from the New York Ophthalmic Hospital. In 1904 he went to Germany and studied under the noted Professor Gruinert of Halle. Upon his return from Europe he relinquished his general practice and started the special practice of eye, ear, nose and throat in Newark, New Jersey. In 1913 he again went abroad and studied in Vienna.

Dr. Beggs was a member of the special staff of the Essex County Homeopathic Hospital in Orange, and a charter member of the organization; a member of the American Academy of Ophthalmology and Otolaryngology; the New Jersey Chiron Club; the Association of Surgeons; the Essex County Medical Society; the American Institute of Homeopathy; the New Jersey State Medical Association. He lived in Glen Ridge, New Jersey, for the past twenty-three years, having his office in Newark. His widow, Margaret H. Beggs, survives him.

Dr. David Lemuel Bettison

Dr. David Lemuel Bettison, Dallas, Texas, died May 9, 1930, in the Methodist Hospital, of injuries received in an automobile accident. He was forty-nine years of age. He graduated from Baylor University College of Medicine, Class of 1906, and had been a teacher of ophthalmology at his Alma Mater for twenty-one years. He was past-President of the Dallas County Medical Society and associate ophthalmologist and otolaryngologist to the Baylor Hospital and Dispensary. He was a member of the American Academy of Ophthalmology and Otolaryngology, and of the American College of Surgeons.

Dr. Oscar Karl Breitenbach

Dr. Oscar Karl Breitenbach, Waukegan, Illinois, died August 14, 1929, of pneumonia, at the age of fifty-one years. He was a graduate of the University of Michigan Medical School, Ann Arbor, class of 1903. He was secretary of the attending staff of the Victory Memorial Hospital; member of the staff of St. Therese's Hospital; consulting oculist and aurist to the Lake County General Hospital. He was a member of the American Academy of Ophthalmology and Otolaryngology, and of the American College of Surgeons.

Dr. Edmund Towle Brown

Dr. Edmund Towle Brown, Burlington, Vermont, died suddenly in June, 1930. Dr. Brown was born on July 18, 1871, in Bridgewater, Grafton County, New Hampshire. In the schools of Bridgewater Dr. Brown secured his earliest academic instruction. Later he attended school at Ashland and Sanbornton, and New Hampton Literary Institute at New Hampton, New Hampshire. He then matriculated at the University of Vermont, where he became a student in the medical department. His entrance into the School of Medicine was in 1895 and he graduated in 1897 with the degree Doctor of Medicine, after which he began practice at Montgomery Center, remaining there until 1908. He then removed to New York City, where he took postgraduate work and became a member of the staff of St. Bartholomew's Hospital under the direction of Dr. Whiting, specialist in mastoid diseases. It followed that he became attached to the staff of Dr. John McCoy of Bellevue Hospital,

New York City, this preceptor being one of the leading surgeons of the eastern metropolis.

In 1908 Dr. Brown went to Vienna for further study of the eye, ear, nose and throat, with the intention of making this field of work his special endeavor, and the following year returned to Burlington. From 1909 to 1916 Dr. Brown was attending oculist and aurist at the Champlain Valley Hospital, Plattsburg, New York; attending clinical surgeon of the eye, ear, nose and throat, Mary Fletcher Hospital, and the Bishop de Goesbriand Hospital; member of the staff of Fanny Allen Hospital, and attending oculist and aurist at the Home for Friendless Women and Home for Destitute Children.

Dr. Brown was a member of the Burlington and Chittenden County Clinical Society, Vermont State Medical Association; Fellow of the American Medical Association; Fellow of the American Academy of Ophthalmology and Otolaryngology; Fellow of the American College of Surgeons; certified by the American Board for Ophthalmic Examinations, and by the American Board of Otolaryngology. His widow and one daughter survive him.

Dr. David Hummell Coover

Dr. David Hummell Coover died January 10, 1930, at his home in Denver, Colorado. His widow, Mary Lenore Coover, whom he married in 1915, survives.

Dr. Coover was born September 29, 1852, the son of Dr. and Mrs. Eli Coover of New Cumberland, Pennsylvania. After finishing the public schools of New Cumberland he entered Jefferson Medical College, where he was one of the youngest students enrolled. At the age of twenty he graduated and began his medical career by interning at the Philadelphia General Hospital, later becoming a general practitioner in the same city. It was not long before he began planning for specialization, and with this in view sailed for Vienna, attending the first course offered in English in the study of the eye, and returning later for more intensive work.

Upon returning to this country Dr. Coover located in Harrisburg, Pennsylvania, but in the Fall of 1899 was obliged to give up his practice there and move to Colorado Springs, because of the failing health of his wife. A year later they took up their

permanent residence in Denver, Colorado, where Mrs. Coover died in 1900, leaving an eight-year-old son, David Hummell, Jr., who also passed away in 1927.

In later years Dr. Coover limited his practice exclusively to diseases of the eye, and became associated with the Gross Medical College. He was made Professor Emeritus of Ophthalmology of the University of Colorado School of Medicine at the time of the reorganization of the medical colleges of Colorado.

Dr. Coover was a member of the American Ophthalmological Society, the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, member of the American Medical Association, Colorado State Medical Association, Colorado Ophthalmological Society, and a charter member of the Denver Clinical and Pathological Society. He belonged to the University Club and the Denver Country Club.

Dr. Coover's success was not alone dependent upon his professional skill, but was furthered by the warm interest and tender sympathy which he held for humanity. His wide circle of personal friends from all walks of life gives evidence of the breadth of his interests.

Dr. William Linsley Dayton

Dr. William Linsley Dayton was born on the 22nd day of March, 1857, at Princeton, Illinois. After completing his studies at the Princeton schools he became a student at Northwestern Medical School, from which he graduated in 1881. He at once took up the study of ophthalmology and otolaryngology, studying in this country and in Europe. His fluent use of the German language made his association with the German clinics very pleasant and made it possible for him to become very closely associated with his instructors. In 1889 he was one of the organizers and patrons of St. Elizabeth's Hospital. He was also at one time lecturer in ophthalmology and otolaryngology at the Lincoln Medical College, the University of Nebraska Medical Department, and the Creighton Medical Department. His kindness and willingness to help his fellow practitioners, and especially younger men in his own specialty, very early earned him the title of "Dean of Ophthalmology of Nebraska."

The outstanding accomplishment in medical development to which Dr. Dayton looked with most pride was his part in the organization of the American Academy of Ophthalmology and

Otolaryngology, he never having missed a meeting from its founding until he was taken sick at the Detroit meeting in 1927. From this illness he gradually declined in health until he died on February 28, 1930, at St. Elizabeth's Hospital. He is survived by one daughter, one son, and two grandchildren.

Dr. Jacob Gray Dorsey

Dr. Jacob Gray Dorsey was born in 1860 and died in 1929. He graduated from the College of Physicians and Surgeons at Keokuk, Iowa, in 1883, and engaged in the practice of general medicine in the territory surrounding Braymer, Missouri, until 1893, at which time he went to Wichita, Kansas, where he limited his practice to diseases of the eye, ear, nose and throat. During the past twenty years he devoted himself exclusively to ophthalmology. He was a pioneer in his specialty and was one of the best known and most loved ophthalmologists in the Midwest. He was very skillful in the operative treatment of cataract, and patients came to him from a wide area to be treated for this affliction.

Dr. Dorsey was a man of the highest moral and ethical principals. He constantly attended the meetings of the County Society and continually endeavored to raise the standards of the medical profession. He was noted for his interest in training young men in his own specialty.

His death from pneumonia on November 15, 1929, followed a short illness of a week, he having carried on his usual heavy practice until he became ill. He will be greatly missed at the meetings of the Academy, which he regularly attended.

Dr. Dorsey's entire life was an unbroken period of assiduous study and work. He was interested only in those things which stood for the betterment of life. It may be truthfully stated that Dr. Dorsey represented the highest type of citizenship and professional standards.

Dr. Wolff Freudenthal

Dr. Wolff Freudenthal was born in Hohensalza, Prussia, January 5, 1858, and attended primary school in that small town. In 1877 he passed his examinations for the "Arbiturium" and entered the University of Breslau. He specialized in botany, anatomy, and physiology. The following year he entered the

University of Leipzig, and later attended the University of Berlin. In October, 1880, he entered the Prussian Army to serve as surgeon and was attached to the Third Guard Regiment.

In 1882 Dr. Freudenthal enrolled in the University of Freiburg in Baden, where he specialized in laryngology and rhinology under Professor Hack, whose introduction and recommendation enabled him to become assistant to Professor B. Fraenkel.

On November 26, 1885, Dr. Freudenthal arrived in New York City, and shortly after was appointed assistant to Dr. Gleitzman at the German Dispensary in the department of Nose, Ear and Throat. The following year he became assistant laryngologist at St. Mark's Hospital.

About this time Dr. Freudenthal became a member of The Academy of Medicine. Twenty years later he was elected Chairman of the Section of Laryngology. He also became an active member of the German Medical Society, acting as its secretary, and in 1896 became President. For many years he served uninterruptedly as a member of the Board of Directors, and again as President.

In 1890 Professor B. Fraenkel invited Dr. Freudenthal to attend the International Medical Congress in Berlin. On his return to America he examined 500 cases of hernia at the Hospital for Ruptured and Crippled, and later wrote an article entitled "The Connection of Abdominal Hernia With Obstructions of the Nose."

Dr. Freudenthal was appointed instructor in the Post-Graduate Medical School and Hospital, and for twenty-eight years served as laryngologist at the Montefiore House. He embodied the results of his work at the latter institution in an article, "The Possibilities of Curing Advanced Laryngeal Tuberculosis." In 1903 Dr. Freudenthal accepted an invitation to read a paper on ozena at the International Medical Congress in Madrid, Spain, delivering the address in Spanish. As a result of his experience as visiting laryngologist at Beth Israel Hospital, he published, in 1894, "Etiology of Pulmonary Tuberculosis," "Nasal Catarrh," and an article on "Colds."

In 1912 the Medical Travel and Study Club of Germany and Austria, under the leadership of Professor His and Professor Albert Oliven, held a meeting at the Liederkranz Club in New York, at which Dr. Freudenthal acted as presiding officer.

Dr. Freudenthal conceived the idea of a Home for Indigent Physicians who had retired from active practice. He presented the plan for such a home to the New York Medical Union, where it was unanimously adopted and a charter obtained.

Dr. Freudenthal succeeded Professor Hans Kudlich as President of the *Gesellig Wissenschaftliche Verein* (German Social Scientific Society of New York), and at the time of his death was again its presiding officer. His many scientific works were published in the leading German, French, English and Spanish medical journals. These were collected in book form, 1887 to 1926. For the last forty-five years he had collected a library on rhinology, otology and laryngology.

Last March, coming from a consultation, he repeated to a colleague the saying of Dr. Oliver Wendell Holmes: "To be seventy years young is sometimes far more cheerful and hopeful than to be forty years old." Ten minutes later he was run over and internally injured. Pneumonia developed, and he died on May 5, 1930, leaving a wife, son and daughter surviving.

Dr. Harold Gifford

Dr. Harold Gifford, ophthalmologist, was born in Milwaukee, Wisconsin, in 1858. He died suddenly on the morning of November 28, 1929, aged seventy-one years.

Dr. Gifford graduated with the degree Bachelor of Science from Cornell University in 1879, and received his medical degree from the University of Michigan in 1882. A year as assistant in the Department of Pathology in the University of Michigan was followed by extensive postgraduate study in several of the European universities. He was appointed first assistant to Horner, then Professor at Zurich and one of the foremost clinic ophthalmologists of Europe. He returned to America and located in Omaha in 1886.

In pure laboratory research, Dr. Gifford's early work on the experimental production of sympathetic ophthalmia, with his demonstration of the fact that organisms may travel from the inoculated eye along the lymph paths into the orbit and thence to the nerve and suprachoroidal spaces of the second eye, and his work on the drainage of the anterior chamber will be remembered.

In bacteriology his contributions have perhaps been most important. He was the first to describe the fact that the normal

conjunctival sac contains numbers of organisms, some of which may become pathologic when they are carried into the eye by trauma or operation. In 1896 he gave the first description in English of the acute conjunctivitis caused by the pneumococcus, and first demonstrated that this organism was the cause, at least in the Middle West, of this disease, by inoculations of secretion containing the organism on his own conjunctiva and that of an assistant, with production of the typical disease. Two years later he published the first description in English of the Morax-Axenfeld bacillus as the cause of chronic conjunctivitis and of corneal ulcer. In 1910 he was the first writer to describe, in English, involvement of the eyelids by the sporothrix, a member of the lower fungi which causes sporotrichosis, and the first in any language to describe a peculiar form of involvement of the ocular conjunctiva with this organism.

As a clinical observer, his name is attached to two eye symptoms which are of value in the diagnosis of exophthalmic goiter, and which he first described in 1906. In 1898 he reported the first American case of Parinaud's conjunctivitis, a rare condition which had previously been described by only a few observers in France and Austria.

In ophthalmic surgery he made many important practical modifications of operations described by others. He devised a form of trachoma forceps for expression of the folds, which presents decided practical advantages over previous instruments, and his brain knife for exploring the brain for abscess, though less known, is a valuable instrument.

Dr. Gifford's writings have appeared chiefly in the form of articles for the various ophthalmologic journals in this country, England and Germany. His most extensive works are the complete reviews of the subjects of sympathetic ophthalmia in the American Encyclopedia of Ophthalmology, and of congenital paresis of the abducens published in part in the American Journal of Ophthalmology. He was one of the editors of the Ophthalmic Record during its existence, and upon its fusion with other journals into the American Journal of Ophthalmology, became a collaborator in that journal.

As an educator, Dr. Gifford was one of the founders of the Omaha Medical College, and served that institution as Professor of Ophthalmology and Otology, and for a time as Dean of the

Faculty. He was one of the most interested in the absorption of that school into the Nebraska University College of Medicine in Omaha, heading the Department of Ophthalmology and Otology in that college when the fusion was effected and until his appointment as Emeritus Professor.

In recognition of his scientific and clinic work he was granted the honorary degree of M. A. by the University of Michigan, and of LL. D. by the University of Nebraska, and was chosen as Chairman of the Section on Ophthalmology of the American Medical Association for its meeting in Cleveland.

His widow, two sons (Dr. Sanford R. and Harold, Jr.), and two married daughters survive.

Dr. Francis C. Howard

Dr. Francis C. Howard of St. Louis, Missouri, died in that city in April, 1930, at the age of thirty-six years. He was born in Fayette, Howard County, Missouri, attending Central College in that town, and graduating in 1914. He later entered Missouri University, acquiring his B. S. degree in 1915.

In 1919 he graduated in medicine at Washington University, St. Louis, and following that had a service in medicine in Johns Hopkins Hospital. In 1919-1920 he had one year on the surgical service of Barnes Hospital. He then became associated with Dr. W. M. C. Bryan in the practice of otolaryngology, until the latter died in 1925, after which Dr. Howard continued the practice alone.

He was instructor in otolaryngology at Washington University School of Medicine, and on the staff of Barnes and St. Louis Children's hospitals.

Dr. John Henry James

Dr. John Henry James, the eldest of three sons of Joseph and Roxanna Caroling Brownell James, was born February 28, 1846, in Washington County, New York, and lived with his parents on a farm, attending district schools with one term at a nearby academy. In February, 1875, he graduated from the University of New York City Medical School. Shortly after graduation he took a position on Blackwell's Island in the Women's Insane Asylum and served for sixteen months. While there he made the acquaintance of Dr. John A. Arnold, afterwards

chosen superintendent of all King County hospitals, and Emil Mayer, then apothecary at the Institute and later the noted laryngologist. In September, 1876, he resigned his position to accept a similar one in the hospital at St. Peter, Minnesota, under the superintendency of Dr. C. K. Bartlett, and served there fifteen years. Resigning in September, 1891, he entered private practice in Mankato, Minnesota, limiting himself to diseases of the eye, ear, nose and throat.

Dr. James was a member of the State Medical Society since 1877; recently made an Honorary member of the Minnesota Valley Association during its existence until it was merged with the Minnesota Southern, of which he became Secretary-Treasurer and later President and Honorary member; charter member of the Blue Earth County Association and also Life Member in same; Fellow in the American College of Surgeons; member of the American Academy of Ophthalmology and Otolaryngology as well as the Minnesota society of the same name.

Dr. James Whittier May

Dr. James Whittier May of Kansas City, Kansas, died December 19, 1929, of pernicious anemia with degeneration of the spinal cord, after a year of illness. He was born in 1879 in Washington Court House, Ohio, the son of the late Doctor James Walker May, who also practiced ophthalmology in Kansas City.

In 1906 he married Miss Lucy Eager, daughter of the late Dr. J. L. B. Eager of Kansas City. Mrs. May, together with two children, survive.

Dr. May was past-President of the Wyandotte County Medical Society, the Kansas Medical Society, the Kansas City Society of Ophthalmology and Otolaryngology, and the Kansas City Southwest Clinical Society. He was also a member of the Kansas City Academy of Medicine, the American Medical Association, the American Academy of Ophthalmology and Otolaryngology, the American College of Surgeons, and the Kansas State Board of Medical Registration, and was on the staffs of Saint Margaret's and Bethany hospitals. During the World War he served overseas on the staff of Base Hospital No. 72 with the rank of Captain. He graduated from the College of Physicians and Surgeons of Kansas City in 1900, and spent his professional life in Kansas City, where he had lived from childhood.

Dr. John Floyd Murdoch

DR. JOHN FLOYD MURDOCH, of Pittsburgh, Pennsylvania, died on January 8, 1930, at the age of sixty years. He was a graduate of the Jefferson Medical College of Philadelphia, class of 1893. He was a member of the American Academy of Ophthalmology and Otolaryngology.

Dr. Walter Eugene Murphy

DR. WALTER EUGENE MURPHY, whose practice was limited to diseases of the eye, ear, nose and throat, was born March 3, 1871, at Cincinnati, Ohio, and died August 3, 1930, at Cincinnati, of cerebral hemorrhage.

Dr. Murphy graduated in 1897 from the Miami Medical College, now the University of Cincinnati School of Medicine. At the time of his death he was President of the Cincinnati League for the Hard of Hearing; also Examining Board of United States Veterans' Bureau (diagnostic center); Chief of Staff, otologic service, Cincinnati General Hospital; Chief of Staff, Children's Hospital; Professor of Otology, University of Cincinnati College of Medicine; was on the staff of the Good Samaritan Hospital and Deaconess Hospital, also Christ Hospital, about 1920.

He served during the World War examining applicants for the Aviation Department, but had no overseas service.

Dr. Murphy was a member of the following societies: American Medical Association, Ohio State Medical Society, American Academy of Ophthalmology and Otolaryngology, Cincinnati Otolaryngological Society, and Cincinnati Academy of Medicine.

Dr. James Allen Patterson

The El Paso County (Colorado) Medical Society lost one of its pioneers November 27th, 1929, in the death of DR. JAMES ALLEN PATTERSON. Dr. Patterson was a former President of the Society and for a score of years had been active in the work of organized medicine.

Dr. Patterson was born November 29, 1859, in Salem, New Jersey. His father, Theophilus Patterson, was also a physician. He obtained his medical degree from Jefferson Medical College, Philadelphia, in 1882. In 1895 he moved to Colorado and obtained his license in that State. He had been a member of the

El Paso County Medical Society for thirty years, and was also a member of the principal ophthalmological societies. He did post-graduate work at Morfields Hospital in London. He married Clara Lippincott in 1886.

Dr. James M. Patton

By the sudden death, June 20, 1930, of Dr. James M. Patton, the medical profession of the State of Nebraska suffered the loss of one of its most prominent members. Dr. Patton was born in Mercersburg, Pennsylvania, on September 25, 1876. When he was six years of age his family moved to Pawnee City, Nebraska, and he received his education there until he attended Bellevue College, and later the Omaha Medical College, now the Medical College of the University of Nebraska, from which he was graduated in 1904. Shortly after graduation he entered the office of Dr. Harold Gifford and became the senior member of the partnership after Dr. Gifford's death last November.

Always interested in his undergraduate college, he was for many years a member of the Board of Trustees of Bellevue College, and at the time of his death was vice-chairman of the Board. He was also a member of the Board of the Presbyterian Theological Seminary of Omaha.

In his scientific life Dr. Patton was pre-eminent. A tireless worker with his patients, who were many, he nevertheless found time to contribute to his branch of the science, ophthalmology, and a rapid search reveals thirty-nine articles on various subjects which he contributed to the literature. These efforts brought him both local and national recognition. He was Professor of Ophthalmology at his Alma Mater, the University of Nebraska; and ophthalmologist to the Nebraska Methodist, Bishop Clarkson Memorial, and Douglas County hospitals. In 1921 he was President of the Omaha-Douglas County Medical Society. He was elected a member of the American Academy of Ophthalmology and Otolaryngology, and the crowning recognition came to him only this year when he received the honor of being elected to membership in the American Ophthalmological Society.

On the rare occasions when Dr. Patton took a vacation from his practice it was nearly always to pursue some further study, or to attend some medical meeting. He had studied in London, Vienna and other foreign centers. At the time of the Great War

he enlisted in the Medical Corps of the Army and was commissioned a Captain. He served overseas with Base Hospital 49, and later in the Hospital Center at Vitel, gaining promotion to the field officer rank of Major.

At the time of his death Dr. Patton was apparently at the height of his efficiency. A few months ago he had been sick a few days in the Methodist Hospital with a bad cold, but apparently had recovered. The day before his death he put in a full day at his office, and in the evening had gone to the Douglas County Hospital. About midnight he was stricken with an attack of angina pectoris and died in less than two hours. His widow and one son survive.

Dr. William Jacob Rideout

Dr. William Jacob Rideout of Freeport, Illinois, died July 25, 1930, at the age of sixty-one years.

He was a graduate of the Keokuk, Iowa, Medical College, class of 1892; was on the staff of the Evangelical Deaconess Hospital. During the World War he served with the Medical Corps. He was a member of the American Academy of Ophthalmology and Otolaryngology, and of the American College of Surgeons.

Dr. Ross Hall Skillern

Dr. Ross Hall Skillern was born November 13, 1875, a son of Samuel Ruff and Sarah Hall Ross Skillern.

His early education was obtained at George F. Martin's school, West Philadelphia, from which he entered the School of Biology, University of Pennsylvania, in 1892, and the Medical School the following year, graduating in 1897. He then began practicing with his father in West Philadelphia and continued until 1903.

After two years he decided to take up the specialty of nose and throat diseases, so went to Vienna, where he worked about one year with Politzer, Hajek, Chiari, and others. He returned in 1907 and opened an office on Chestnut street.

Finding that the younger men showed great interest in the nasal sinuses, Skillern began giving courses on operations on these cavities on the cadaver, being greatly aided by the University authorities. These courses were continued for several years until he was called to the faculty of the Medico-Chi as Professor

of Laryngology. During this period he made original researches in the ethmoid sinuses, the work being carried out in the McManes Laboratory, University of Pennsylvania, and wrote a textbook on the Accessory Sinuses of the Nose which has become the standard work on this subject in the English language.

About this time the Philadelphia Laryngological Society was organized and he was chosen the first President, later being elected an honorary Life Member.

When war was declared with Germany, Dr. Skillern was one of the first of the specialists to join the colors, having received a request from the Surgeon-General's office to take command of a head unit for overseas duty. He was commissioned Major and sent to Camp Sheridan, Montgomery, Alabama, in charge of the division of head surgery. After organizing this Department and putting it on a sound, functioning basis, Dr. Skillern was promoted to Lieutenant-Colonel and put in charge of a base hospital and sent to France, where he was stationed at Mesves-sur-Loire until after the Armistice.

On returning home he took up his duties with the Graduate Hospital as Professor of Laryngology, a position he held until his death.

Dr. Skillern was much interested in national societies in his specialty and was elected Chairman of the Section on Otolaryngology of the American Medical Association in 1920, and First Vice-President of the American Academy of Ophthalmology and Otolaryngology in 1921. He was a member of the National Board of Examiners in Otolaryngology in 1923; First Vice-President of the American Laryngological Association in 1925; President of the American Academy of Ophthalmology and Otolaryngology in 1927, and President of the American Laryngological, Rhinological and Otological Society in 1930. He was invited to be the Guest of Honor at the meeting of the British Medical Association, held in Cardiff, Wales, in July, 1928.

Dr. Skillern was a member of the Union League, University Club, Meriod Cricket and Golf Club, Physicians' Motor Club, Medical Club, Manufacturers' Club, Automobile Club, Surgeons' Club of Rochester, Historical Society, Main Line Civic Association, American Medical Association, American Laryngological Association, American Triological Society, American College of Surgeons, American Academy of Ophthalmology and Otolaryngology, Philadelphia Laryngological Society, Kansas City Eye,

Ear, Nose and Throat Society, honorary member Medical and Surgical Society of Washington, D. C., honorary member Pacific Coast Otolaryngological Society, honorary Fellow American Stomatological Association, member American Medical Editors' Association, and the College of Physicians (Philadelphia).

Dr. Skillern is survived by his widow and four children.

Dr. S. MacCuen Smith

Dr. S. MacCuen Smith, after a brief illness from angina pectoris, died on September 14, 1929, at the age of sixty-six.

He was born in Blair County, Pennsylvania, in 1863. He received his early education at Hollidaysburg Academy, and graduated from Jefferson Medical College in 1884. Early in his medical career he became especially interested in diseases of the ear, nose and throat, and when the Germantown Hospital established a department of Otolaryngology, Dr. Smith was placed in charge.

He was appointed Professor of Otology at Jefferson Medical College in 1904. This position he filled with great honor to the College and to himself until his death.

Among his other professional connections Dr. Smith was attending otologist, Jefferson Medical College Hospital; otolaryngologist, Germantown Hospital; aurist and laryngologist, Jewish Hospital; consulting aurist, American Oncologic Hospital, Philadelphia; Memorial Hospital, Roxborough; State Hospital of Nanticoke, Nanticoke. He was a Fellow of the American Otological Society; American Laryngological, Rhinological and Otological Society; American Academy of Ophthalmology and Otolaryngology; American Medical Association; American College of Surgeons; and the College of Physicians of Philadelphia.

Dr. Smith served as Chairman of the Section on Laryngology, Otology and Rhinology, American Medical Association, 1907; President American Laryngological, Rhinological and Otological Society, 1916. He was made President-elect of the American Otological Society at its meeting in June, 1929.

Dr. Smith was editor of the American edition of the "Atlas and Epitome of Otology." He contributed a chapter on "The Treatment of Diseases of the Ear and Tympanic Membrane" in the volume "Modern Treatment" by American and English authors. He was also author of a chapter, "Diseases of the Middle Ear," in Sajou's "Analytic Cyclopedia of Practical Medicine." In

addition, he contributed a chapter on "Indications for Surgery of Mastoid Process" in a volume, "The Nose, Throat and Ear and Their Diseases," edited by Chevalier Jackson and George Morrison Coates. Numerous other writings have appeared in various journals from time to time.

His widow, two sons and a daughter survive.

Dr. Louise Vandervoort Stegman

Dr. Louise Vandervoort Stegman was born in New Antioch, Ohio, August 7, 1868. Her early womanhood was passed in Dayton, Ohio. She was graduated from the American Medical Missionary College in Battle Creek, Michigian, in 1906. After a brief period in private practice in Missoula, Montana, she entered the service of the Battle Creek Sanitarium, to continue there to the end.

Dr. Stegman devoted herself to ophthalmology, becoming the head of the Eye Department of the Sanitarium. For eight years she was also the obstetrician, having prepared herself for this work by a course in the Lying-In Hospital in Philadelphia.

She took a number of postgraduate courses. Some of these were at the Cornell Medical School and the New York Eye and Ear Infirmary. When Dr. Ernst Fuchs of Vienna held a class in Chicago, she attended it. At his suggestion she went to Vienna for study in 1922-1923. She also had some instruction in Paris and London. In 1926 she assisted at the great eye clinic which is held each year at Shikarpur, India. More than a thousand cataract operations were done there, of which she performed two hundred forty-six, besides doing other surgery. In Delhi and Madras, India, she likewise attended clinics. She made this journey with her husband, Henry M. Stegman, to whom she had been married in 1913. They returned by way of China and Japan, thus making a world tour.

One year Dr. Stegman accompanied Dr. J. A. Stucky of Lexington, Kentucky, as a volunteer worker in his series of clinics held in the Kentucky mountains.

She held the certificate of the American Board of Ophthalmologic Examiners, and was a member of the Academy of Ophthalmology and Otolaryngology. She attended the national gatherings of ophthalmologists as often as possible, sometimes taking part in the discussion of papers. She wrote various papers her-

self, the most important being based upon her research into the connection between certain eye manifestations and thyroid imbalance.

In 1920 Dr. Stegman was elected a member of the Electoral College which formally chose Harding as President.

While attending the opening of the Wilmer Eye Clinic at Johns Hopkins, she was stricken with an illness which at first seemed to be appendicitis. It was, however, cancer of the ileum, as an operation revealed. She was taken to California to try the Coffey-Humber treatment, and died in Los Angeles, March 23rd last.

Dr. Frederic M. Strouse

Dr. Frederic M. Strouse was born in San Francisco, California, June 23, 1864. He was educated in the common schools of that city until he reached the age of nine, when his father removed to St. Paul, Minnesota. The boy's further education took him through St. Paul High School. Upon his graduation from high school Dr. Strouse entered the Medical School of the University of Pennsylvania, class of 1885. Classmates have said that he was a student of much distinction.

Dr. Strouse opened his first office in Shamokin, Pennsylvania, but after a brief time removed to Philadelphia, where he entered upon general practice and joined the staff of the Stetson Dispensary. During the aftermath of the Johnstown flood he and other surgeons from the Dispensary were on the scene administering to the injured and sick, having been dispatched there at the instance of the late John B. Stetson. Somewhat more than forty years ago he became medical examiner for the Northwestern Mutual Life Insurance Company, a post he held up to the time of his death.

On August 3, 1892, Dr. Strouse married Miss Mattie Pollock of Philadelphia, who survives him. They had no children.

Dr. Strouse remained in general practice for more than twenty years. In 1914 he studied under the late Professor Ross Hall Skillern, and then specialized in laryngology. He was one of the founders of the Philadelphia Laryngological Society and became its third President. He was also a Fellow of the College of Physicians, and was secretary of the Laryngological Section at the time of his death.

In 1919 Dr. Strouse was appointed Instructor of Laryngology and Rhinology at the Post-Graduate Hospital. In 1921 he became Assistant Professor, and was elevated to Associate Professorship four months before his death. He was also Chief of the Laryngological Dispensary.

Other organizations with which he was identified are: The American Medical Association, Philadelphia County Medical Association, Medical Club, American Academy of Ophthalmology and Otolaryngology, and the Glassboro, New Jersey, lodge of Masons.

Dr. Strouse was struck by an automobile in Atlantic City, on August 4, 1930, and died one hour later in the Atlantic City Hospital.

Dr. Derrick Tilton Vail

Dr. Derrick Tilton Vail was born in Franklin, Warren County, Ohio, on October 2, 1864, and died in Cincinnati, Ohio, October 29, 1930.

He attended the public school; took the four years' course at the Franklin Union High School, and graduated in 1883, valedictorian of his class, at the age of nineteen years. He then taught school at Franklin and Miamisburg, Ohio, for five years, during which time he studied medicine under the guidance of Dr. Richard P. Evans, of Franklin.

He attended the Miami Medical College, Cincinnati, Ohio, (Medical Department of the University of Cincinnati), and graduated with an M. D. degree in 1890. He was the Class Orator. While an undergraduate he won the Robert Sattler first prize in ophthalmology.

Later he associated himself with Dr. Christian R. Holmes, of Cincinnati, and remained with him for ten years in the practice of ophthalmology and otolaryngology.

In 1899, 1909, and 1911, Dr. Vail visited clinics in London, Hamburg, Zurich, Berlin and Vienna, and made two trips to India in 1909 and 1924. After his return from India in 1910, he began to write and work extensively upon the cataract operation.

Perhaps his most outstanding contribution to ophthalmology was his discovery, in 1913, and report in 1914, of an entirely new disease of the eye, which he named "Squirrel Plague Conjunc-

tivitis," or "Bacillus Tularense Infection of the Eye." It proved to be the first case of Tularemia in man diagnosed and proved bacteriologically. The laboratory tests, bacteriologic investigation, animal experimentation, and diagnosis, were made by William B. Wherry, Professor of Bacteriology of the College of Medicine, University of Cincinnati.

Dr. Vail was Clinical Professor of Ophthalmology in the Laura Memorial College, and Staff Ophthalmologist to the associated Presbyterian Hospital, Cincinnati, 1895-1903; Clinical Professor of Ophthalmology in the Miami Medical College from 1896 until 1909, when it merged with the Ohio Medical College to form the College of Medicine of the University of Cincinnati, at which time he was made Chief of the Eye Clinic, and continued Professor of Ophthalmology until 1912, when he was made Professor Emeritus of Ophthalmology of the College of Medicine, University of Cincinnati.

He was a founder member of the following societies and institutions:

The American Academy of Ophthalmology and Otolaryngology (President in 1908), The Oxford (England) Ophthalmological Congress (1909) (Member of the Council), The American College of Surgeons (1913), The Gorgas Memorial Institute (1916).

He was awarded a certificate, without examination, in 1917, by the American Board of Ophthalmic Examiners.

He was an honorary member of the American Laryngological, Rhinological and Otological Society; a member of the American Ophthalmological Society, The Chicago Ophthalmological Society, American Medical Association (Ophthalmic Section), The Ohio State Medical Association (Eye, Ear, Nose and Throat Section) of which he was Chairman in 1921, the Cincinnati Academy of Medicine (Trustee in 1922), The Cincinnati Ophthalmological Club (President in 1919).

Dr. Vail was forced to retire from active practice in January, 1928, after an attack of influenza, complicated by myocarditis. His last article was, "The Intracapsular Versus the Capsulotomy Cataract Operation." (The Sind Medical Journal, Karachi, India, Apl. 1928.)

He is survived by his widow, Della Harris Vail; two sons, Doctors Harris H. Vail and Derrick T. Vail, Jr., and two daugh-

ters, Mrs. Harold S. Dwight of Cincinnati, and Mrs. Melville Borders of Kansas City, Missouri.

Dr. Lee M. Willard

Dr. Lee M. Willard, of Wausau, Wisconsin, died November 14, 1929, at a hospital in Cleveland, Ohio, at the age of sixty-one years.

He was a graduate of the College of Physicians and Surgeons of Chicago, class of 1891. He was a member of the American Academy of Ophthalmology and Otolaryngology, and of the American College of Surgeons. He was on the staff of St. Mary's Hospital, and the Wausau Memorial Hospital.

In memory of the departed the members stood in silence for one minute.

It was moved by Dr. George F. Suker that a telegram of condolence be sent to Mrs. Vail, word having been received of the death of Dr. Derrick T. Vail Tuesday night. Motion seconded and carried.

UNFINISHED BUSINESS

THE PRESIDENT: Under this heading we first take up a very important matter—a change in the Constitution and By-Laws. If you have your programs you may read the proposed change in Article IV, as follows. This will be read by the Secretary:

ARTICLE IV

The officers of this Academy shall be:

President
President-Elect
First Vice-President
Second Vice-President
Third Vice-President
Comptroller
Executive Secretary and Treasurer
Secretary for Ophthalmology
Secretary for Otolaryngology
Secretary for Instruction
Associate Secretary for Instruction
Editor of Transactions
Board of Councillors

These officers shall be elected annually, except the Councillors, who are to serve four years, one to be elected each year.

The Council shall consist of the President, who shall be the chairman; President-elect, First Vice-President, the three most recent past Presidents, and the four active Fellows who shall have been elected to serve four years. The Comptroller and the Executive Secretary shall be ex-officio members of the Council.

Nominations for officers are presented to the Academy by the Council. Other nominations may be made by a written petition signed by five (5) active Fellows.

The nominations shall be made and the election held at an annual meeting. A majority vote cast by the active Fellows present at such a meeting shall constitute an election.

BY-LAWS

ARTICLE IV

Section 4: The Comptroller shall be the financial officer of this Academy, representing and responsible to the Council. He

shall approve all budgets, supervise the work done in the Executive Secretary-Treasurer's office, sign all vouchers with the Executive Secretary and Treasurer. He shall arrange, in conjunction with the Finance Committee, all investments and shall in general be responsible for the expenditures of all moneys. He will provide a suitable bond and be allowed an honorarium by the Council.

Section 5: The Executive Secretary and Treasurer will conduct the business affairs of the Academy, collect dues, draw, sign and send all vouchers for payment of indebtedness to the Comptroller for approval. He, with the approval of the Comptroller and Finance Committee, shall arrange with a proper bank or trust company for the safe keeping of all moneys and investments belonging to the Academy. He shall so keep the books of this Academy that a monthly audit can be sent to each member of the Council and the Comptroller. He shall be held responsible for the work done by each of the subofficers. He shall have the privilege of nominating, subject to the approval of the Council, the different secretaries of sections. He will provide a suitable bond and be allowed an honorarium.

DR. A. J. BEDELL: I move the adoption of these amendments. (Motion seconded by Dr. Gradle and carried.)

NEW BUSINESS

DR. S. J. KOPETZKY: For the benefit of those who did not attend the meeting held this evening by those interested in teaching, I would like to say that it was the sense of that meeting that this resolution should be presented here for consideration.

WHEREAS, those of our members engaged in teaching the specialties of otolaryngology and ophthalmology have neither a forum for the exchange of ideas nor the means by which to supervise and when necessary standardize the courses they give, and as a group are inarticulate; and

WHEREAS, it has seemed to us that the formation of a Section on Teaching would supply a need felt by many; therefore

BE IT RESOLVED, that the Council be instructed to appoint the necessary officers or committeemen to form such a section, the rules and regulations adopted and approved by the Council for the administration of such a section; and

BE IT FURTHER RESOLVED, that the proper authorities shall enact the necessary measures to provide the finances needed in the formation and functioning of such section.

ARTHUR J. BEDELL
ARTHUR W. PROETZ
L. W. DEAN
J. A. STUCKY
SAMUEL J. KOPETZKY
RALPH A. FENTON
FRANK E. BURCH
T. E. CARMODY
G. M. COATES

I move the adoption of this resolution. (Motion seconded and carried.)

Election of Members

THE SECRETARY: The following applications for membership have been approved by the Council:

Agan, William Byron, 510 Third Street, Brooklyn, N. Y., Op.
Baldwin, Herschel E., 403-5-7 Temple Bldg., Danville, Ill., ALR.
Bassen, Edward J., 698 Madison Ave., New York, N. Y., Op.
Baum, William Wells, Bank of Commerce Bldg., Salem, Oregon, ALR.
Beals, C. Wearne, 41 N. Brady St., DuBois, Pa., ALR.
Bender, Henry A., 721 Black Bldg., Waterloo, Iowa, ALR.
Bender, Morris Samuel, 11 150th St., Jamaica, N. Y., ALR.
Bernheimer, Benno L., 104 S. Michigan, Chicago, Ill., ALR.
Berry, Gordon, 36 Pleasant St., Worcester, Mass., ALR.
Boemer, Lilburn Casper, 3720 Washington Blvd., St. Louis, Mo., ALR.
Bergeson, Egbert John, 1005 Yates Bldg., Minneapolis, Minn., Op.
Bothman, Louis, 122 S. Michigan, Chicago, Ill., Op.
Bruce, Gordon Murphy, 180 Fort Washington Ave., New York, N. Y., Op.
Burns, Louis J., 1930 Chestnut St., Philadelphia, Pa., ALR.
Cady, Donald W., 65 North Madison Ave., Pasadena, Cal., Op.
Campbell, Angus Alexander, 86 Bloor St., W., Toronto, Canada, ALR.
Carpenter, Chapin, 1930 Chestnut St., Philadelphia, Pa., Op.
Cary, Edward Fayette, 171 West 57th St., New York, N. Y., ALR.

Chandler, Gail Ellsworth, 502 Huntington Bldg., Miami, Fla., ALR.

Clement, Lucian Orville, Bank of Commerce Bldg., Salem, Ore., OALR.

Cohoe, Don A., 13535 Woodward Ave., Highland Park, Detroit, Mich., ALR.

Conway, John Moore, 5151 Jenkins Arcade, Pittsburgh, Pa., ALR.

Cortopassi, Andre John, 704 Second Nat. Bank Bldg., Saginaw, Mich., ALR.

Corya, Harold W., 30 E. 40th St., New York, N. Y., ALR.

Cummings, George Otis, 47 Deering St., Portland, Maine, ALR.

Cutino, Rodolph M., 165 Clinton St., Brooklyn, N. Y., Op.

Davies, Harry Francis, 1835 Eye St., N. W., Washington, D. C., ALR.

Davis, William C., 327 E. State St., Columbus, Ohio, Op.

Day, Kenneth Mosier, 121 University Place, Pittsburgh, Pa., ALR.

Dechter, M. Archibald, Professional Bldg., Westfield, Mass., ALR.

Depping, Charles William, 17 West 54th St., New York, N. Y., ALR.

Dobbs, Otto Ross, 2435 N. Grand Blvd., St. Louis, Mo., ALR.

Eagleton, Wells Phillips, 15 Lombardy St., Newark, N. J., ALR.

Ebeling, William Bruno, 54 Greene Ave., Brooklyn, N. Y., Op.

Fackler, Charles Lewis, 11 E. Market St., York, Pa., ALR.

Findlay, Ephraim K., Suite 904, 30 N. Michigan Ave., Chicago, Ill., Op.

Fineberg, Maxwell, 3858 Westminster, St. Louis, Mo., ALR.

Gabriel, Carson King, Western Catholic Union Bldg., Quincy, Ill., ALR.

Gilliland, Oliver Sheley, 801 Shukert Bldg., Kansas City, Mo., ALR.

Gouterman, Joseph I., 1607 W. Diamond St., Philadelphia, Pa., Op.

Grant, Hendrie Walter, Hamm Bldg., St. Paul, Minn., Op.

Greene, Maurice Lytton, 4178 Flad Ave., St. Louis, Mo., Op.

Greenwood, Glenn Jackson, 317 First Ave., East, Newton, Iowa, ALR.

Griffith, Harold M., 804 Johnstown Trust Bldg., Johnstown, Pa., Op.

Hahn, William Horace, 15 Lombardy St., Newark, N. J., Op.
Hampsey, Alexander Richard, 806 May Bldg., Pittsburgh, Pa., ALR.
Hardie, George Christopher, 290 W. Michigan Ave., Jackson, Mich., ALR.
Harris, Edward Williams, 681 E. Broad St., Columbus, Ohio, ALR.
Heetderks, Dewey Ralph, 405 Medical Arts Bldg., Grand Rapids, Mich., ALR.
Helfrich, Edward Daniel, 269 E. State St., Columbus, Ohio, ALR.
Henderson, Lawrence Edward, 168 Sterling St., Watertown, N. Y., Op.
Hickey, Harold Lowry, 934 Republic Bldg., Denver, Colo., ALR.
Hicks, William Joseph, 15 King St., Middletown, N. Y., ALR.
Hilgartner, Henry Louis, 209 Norwood Bldg., Austin, Texas, Op.
Hodge, George Esplin, Medical Arts Bldg., Montreal, Canada, ALR.
Hooss, Charles Henry Albert, 3115 South Grand, St. Louis, Mo., Op.
Hopkins, Guy Huskinson, 702 N. Main St., Pueblo, Colo., Op.
Hunter, Robert John, 2011 Chestnut St., Philadelphia, Pa., ALR.
Hutchison, Keith Ogilvie, 1509 Sherbrooke St. W., Montreal, Canada, ALR.
Jones, Marvin Fisher, 121 East 60th St., New York, N. Y., ALR.
Judd, John H., Medical Arts Bldg., Omaha, Neb., Op.
Keiper, George Frederick, Jr., 727 West 7th St., Los Angeles, Calif., ALR.
Kline, Oram R., 414 Cooper St., Camden, N. J., ALR.
Knauer, William Jerome, 33-36 Buckman Bldg., Jacksonville, Fla., ALR.
Kreutz, George Charles, Henry Ford Hospital, Detroit, Mich., ALR.
Landis, James Everhart, 517 Walnut St., Reading, Pa., ALR.
Leech, Vernon Mayne, 55 E. Washington St., Chicago, Ill., Op.
Leavenworth, Richard Ormond, 836 Lowry Med. Arts Bldg., St. Paul, Minn., Op.
Lillie, Walter Ivan, Mayo Clinic, Rochester, Minn., Op.
Livingston, George Shaynin, 185 N. Wabash Ave., Chicago, Ill., ALR.
Lukens, John Alfred, 316 Michigan St., Toledo, Ohio, ALR.
Lurie, Moses H., 483 Beacon St., Boston, Mass., ALR.

McCormick, Thomas Francis, 120 E. Wisconsin Ave., Milwaukee, Wis., ALR.
McCoy, Leslie Layton, 817 Summit Ave., Seattle, Wash., Op.
McGillicuddy, Oliver B., University Hospital, Ann Arbor, Mich., ALR.
McHenry, Lawrence Chester, 117 N. Broadway, Oklahoma City, Okla., ALR.
McRae, John Hunter, Grand Rapids Clinic, Grand Rapids, Mich., Op.
MacDonald, Joseph C., 301 West 12th St., Oklahoma City, Okla., ALR.
Mann, Benjamin H., 2208 S. Broad St., Philadelphia, Pa., Op.
Marsden, Wilson Conrad, 64 N. Franklin St., Wilkes-Barre, Pa., ALR.
Martin, William Owen, Jr., 412 Medical Arts Bldg., Atlanta, Ga., Op.
Means, Russell Garrett, 137 East State St., Columbus, Ohio, ALR.
Merica, Forrest Wayne, 14805 Detroit Ave., Lakewood, Ohio, ALR.
Miller, H. Edward, 728 Missouri Bldg., St. Louis, Mo., ALR.
Miller, John, 121 E. 60th St., New York, N. Y., ALR.
Mitchell, Horace Ervin, 15701 Detroit Ave., Lakewood, Ohio, ALR.
Moore, Paul Guthrie, 1701 Medical Arts Bldg., Cleveland, Ohio, Op.
Moore, Paul McNary, Jr., University Hospital, Iowa City, Iowa, ALR.
Moore, Robert Booth, Trinity Hospital, Little Rock, Ark., ALR.
Munson, Frederick TenEycke, 2201 E. Jefferson Ave., Detroit, Mich., ALR.
Murphy, Albert Bernard, 2830 Grand Ave., Everett, Wash., ALR.
Nelson, Leo Arthur, 4105 Live Oak St., Dallas, Texas, ALR.
Nugent, Oscar Benton, 231 W. Washington St., Chicago, Ill., Op.
O'Brien, Gerald Richard, 307 Sterling Place., Brooklyn, N. Y., ALR.
Olmstead, William DeF., 1920 Pacific Ave., Atlantic City, N. J., Op.
Pace, Errett, 711 City National Bank Bldg., Paducah, Ky., ALR.
Paul, William C., Washington Bldg., Tacoma, Wash., ALR.
Payne, Virgil LeRoy, 1018 Madison Ave., Memphis, Tenn., ALR.
Persky, Abram H., 1922 Spruce St., Philadelphia, Pa., ALR.

Porter, Charles Terrell, 520 Commonwealth Ave., Boston, Mass., ALR.

Pope, Ernest Clark, 1931 First Ave., Birmingham, Ala., ALR.

Postle, Carlton David, 240 East State St., Columbus, Ohio, ALR.

Potter, Willis A., 1355 David Whitney Bldg., Detroit, Mich., ALR.

Preston, Henry Grant, Professional Bldg., Harrisonburg, Va., ALR.

Reed, William Wallace, Mills Bldg., Topeka, Kansas, ALR.

Reid, Horace Withers, 19 Garfield Place, Cincinnati, Ohio, Op.

Rubenstein, Asher Harry, Medical Arts Bldg., Syracuse, N. Y., ALR.

Ryan, Edward R., 425 East Water St., Milwaukee, Wis., Op.

Sebert, Louis Joseph, 506 Medical Arts Bldg., Toronto, Can., Op.

Shapiro, Sherman Lawrence, 25 E. Washington St., Chicago, Ill., ALR.

Simpson, Morris Bigelow, 720 Rialto Bldg., Kansas City, Mo., ALR.

Simons, Neal Samuel, MacCormack Clinic, Whitehall, Wis., ALR.

Sloane, Henry Oscar, 1717 Pine St., Philadelphia, Pa., Op.

Smith, William Leroy, 1005 Medical Arts Bldg., Salt Lake City, Utah, Op.

Stanley, Court Roger, Worthington, Minn., ALR.

St. Clair, Charles Tiffany, Bluefield, W. Va., ALR.

Stone, Harry B., 702 Shenandoah Bldg., Roanoke, Va., ALR.

Tebbutt, Harry Knibb, 240 State St., Albany, N. Y., ALR.

Theobald, Walter Henry, 307 N. Michigan Ave., Chicago, Ill., ALR.

Thomashefsky, Milton, 135 Eastern Parkway, Brooklyn, N. Y., ALR.

Tillotson, Rulon Squires, Woodland Clinic, Woodland, Calif., ALR.

Tomb, Everett Hale, 110 E. Wisconsin Ave., Milwaukee, Wis., ALR.

Uhler, S. Mann, 104 North 8th St., Allentown, Pa., ALR.

Van Dellen, Alfred Lubbert, 6857 S. Halstead, Chicago, Ill., Op.

Van Horn, Alfred Fellows, 514 Central Ave., Plainfield, N. J., ALR.

Ward, James W., Exchange Bldg., Greenville, Texas, ALR.

Watkins, Harold R., Griesheim Bldg., Bloomington, Ill., ALR.

Webster, Franklin Raymond, 720 State Tower Bldg., Syracuse, N. Y., Op.

Weeth, Charles Reed, 96 Joralemon St., Brooklyn, N. Y., ALR.

Weidlein, Ivan Ford, 614 Osborn Bldg., Cleveland, Ohio, ALR.

Wetzel, John Otto, 404 Capitol Saving and Loan Bldg., Lansing, Mich., Op.

Wexler, David, 55 East 86th St., New York, N. Y., Op.

White, Orton Eugene, 28 N. Main St., Cortland, N. Y., ALR.

Wilkinson, Arthur Paul, 664 Fisher Bldg., Detroit, Mich., ALR.

Wilkinson, Oscar, 1408 L St., N. W., Washington, D. C., OALR.

de Yoanna, Alfred Aurelius, 111 Pierrepont St., Brooklyn, N. Y., ALR.

Burritt, Norman W., 30 Beachwood Rd., Summit, N. J., OALR.

Carter, Earl Dwight, 603 Beacon Bldg., Wichita, Kansas, OALR.

Dillon, Ira Hugh, Hatcher Hospital, Wellington, Kansas, OALR.

Hunter, James Finlay, 1737 Chestnut St., Philadelphia, Pa., ALR.

Joynt, Martin John, Royal Bldg., LeMars, Iowa, OALR.

Kuhn, Hedwig Stieglitz, First Trust Bldg., Hammond, Ind., Op.

Laff, Herman I., 406 Metropolitan Bldg., Denver, Colo., ALR.

Lingeman, Byron Newton, 419 Ben Hur Bldg., Crawfordsville, Ind., OALR.

Malone, Frank B., Lubbock Sanitarium, Lubbock, Texas, ALR.

Morley, George Alfred, Crookston State Bank Bldg., Crookston, Minn., OALR.

Oppegaard, Chester Leonard, 220 S. Broadway, Crookston, Minn., OALR.

Rigg, James Paul, Clinic Bldg., Grand Island, Neb., OALR.

Robison, James Thomas, 309 State St., New London, Conn., ALR.

Schluer, Elmer Pfingsten, 820 Metropolitan Bldg., St. Louis, Mo., ALR.

Stonecypher, David Daniel, 107 S. 9th St., Nebraska City, Neb., ALR.

Toot, Joseph Frederick, 923 First National Bank, Canton, Ohio, Op.

Wenner, Waldemar Thomas, 303 St. Mary's Bldg., St. Cloud, Minn., OALR.

Whitney, Elmer Lemuel, Henry Ford Hospital, Detroit, Mich., OALR.

I move that this list of 154 applicants be elected to membership. (Motion seconded and carried and the applicants declared elected.)

Election of Officers

DR. HARRIS P. MOSHER: The Nominating Committee reports to Council, and Council reports to you, as is their duty, the following names for officers, as called for by the Constitution:

President	Dr. John F. Barnhill	Miami Beach, Fla.
President-Elect	Dr. S. Hanford McKee	Montreal, Canada
1st Vice-President	Dr. Meyer Weiner	St. Louis, Mo.
2nd Vice-President	Dr. John W. Cormack	Indianapolis, Ind.
3rd Vice-President	Dr. Ben Witt Key	New York City
Comptroller	Dr. Secord H. Large	Cleveland, Ohio
Executive Secretary-Treas.	Dr. William P. Wherry	Omaha, Neb.
Secretary for Ophthalmology	Dr. William L. Benedict	Rochester, Minn.
Secretary for Otolaryngology	Dr. John L. Myers	Kansas City, Mo.
Secretary for Instruction	Dr. Harry S. Gradle	Chicago, Ill.
Associate Secretary	Dr. William V. Mullin	Cleveland, Ohio
Editor Transactions	Dr. Arthur W. Proetz	St. Louis, Mo.
Associate Editor	Dr. Lawrence Post	St. Louis, Mo.
Member of Council	Dr. Ralph A. Fenton	Portland, Ore.
Member Board of Ophthalmic Exam.	Dr. William H. Wilder (to succeed Dr. Patton)	Chicago, Ill.

DR. ROBERT L. FORGRAVE, St. Joseph, Missouri: I move that this report be accepted and the Secretary be instructed to cast the ballot of the Academy for the officers named. (Motion seconded and carried and ballot cast by Secretary.)

Place of Meeting

DR. HARRIS P. MOSHER: The place of meeting for 1931 will be French Lick Springs, Indiana.

THE PRESIDENT: I have no "swan song" to sing, but I have the very pleasant duty of assisting in the induction of your newly-elected President into office. I will ask Dr. Suker and Dr. Felt to serve as a committee to escort Dr. Barnhill to the Chair.

Dr. John F. Barnhill: I assure you, ladies and gentlemen, that I have no speech to make. However, I should be decidedly inhuman should I not feel a sense of very deep gratitude toward the Fellows of the greatest Association in the world devoted to the study of eye, ear, nose and throat. I have for more than a third of a century been a member of this Association. I have seen it grow from a membership of about twenty-five to almost two thousand; I have seen its activities increase from a few papers that were fairly scientific to a sufficient number of papers that are ultra scientific; I have seen it grow from taking in two or three members a year, to 154 members who come in after a most rigid scrutiny and most careful examination.

We have a hard task to perform next year. We have a difficult proposition before us—to beat, as it were, the greatest convention in the history of this Academy, which has just been held under the leadership of our honored President, Dr. Wilder. We will not make progress unless we do beat that record, and we are going to do it. The reason I say that we are going to do so is that we have the same secretaries that we had last year and their experience is increasing.

Just a word about the place of meeting. It is one of the most beautiful spots in America. The convention will be held in one of the best hotels in America. It is located within a few miles of the center of population of the United States, and is easily accessible. All the Hoosier populace knows you are coming, and you will be welcomed by a hospitality such as you have almost never known. I wish you, therefore, to mark down immediately in your appointment book an engagement to come to French Lick Springs, Indiana, the middle of next September, and bring your wives and daughters with you.

The Business Session of the American Academy of Ophthalmology and Otolaryngology adjourned.

Report of Committee on Ophthalmic and Oto-Laryngic Pathology

In 1922 there was established at the Army Medical Museum a special department of Ophthalmic and Oto-Laryngic Pathology. This department was initiated by the American Academy of Ophthalmology and Oto-Laryngology. Later, the American Ophthalmological Association and the Ophthalmological Section

of the American Medical Association joined in the maintenance of the collection. The object was to obtain large numbers of specimens for the purpose of study and research. During the past five years the American Academy has financed the making of twelve sets of 100 slides each illustrating the pathology of the eye. Thus, the use of the collection as an educational measure has been included in the activities of the department.

After numerous specimens of tumors of the eye were received the department began a follow-up system on those cases, the outcome of which would be of value. In this way the department has acted as a registry of ophthalmic pathology. Other registries have been established at the Army Medical Museum, the Lymphatic Tumor Registry of the American Association of Pathologists and Bacteriologists in 1925, and the Bladder Tumor Registry of the American Urological Association in 1927. These registries are now well established and have reached that stage of development and activity which makes necessary more professional, technical and clerical work than can be given by the Army Medical Museum.

That adequate support might be obtained and that a continuous policy be carried out, the project was presented to the Division of Medical Science of the National Research Council by the chairman, Dr. Ludwig Hektoen, and a committee was formed by that council for the "American Registry of Pathology." The committee is composed of the following: Dr. Howard T. Karsner, chairman; Dr. Bowman C. Crowell, Dr. James Ewing, Dr. Harry S. Gradle, Dr. Herman L. Kretschmer, Dr. Stanley P. Reimann and George R. Callender, secretary. These men have been active in promoting the registries already established, or were included because of their membership in associations naturally interested, as the American Association for Cancer Research and the American Society for Cancer Control.

The registry as contemplated will require an annual budget of over $15,000 per annum, and it is hoped to obtain an endowment to cover that amount. It is important that those societies having a special interest indicate their approval of this plan to finance the registries and place them on the secure and permanent basis they require.

It is realized that the annual budget of the American Registry of Pathology cannot be met from society contributions, but some of the societies are in position to help. It is believed that

a contribution, no matter how small, will add significance to any resolution approving the formation of the American Registry of Pathology.

The Committee on the American Registry of Pathology asks that the American Academy of Ophthalmology and Oto-Laryngology consider this proposal and hopes that the Academy will by resolution formally indorse the registry and make such contribution to it as it deems possible.

This indorsement by interested medical societies will be of the greatest value in presenting the proposition to those who may be able to contribute in a larger way to the permanent support of the registry. It is urged that action on it be taken at the coming meeting of the Academy.

There follows an outline explaining the object and scope of the registry more fully than does this letter.

For the committee:

(Signed) GEO. R. CALLENDER, Secretary.

AMERICAN REGISTRY OF PATHOLOGY

The object of this registry is to collect data and specimens from patients, especially those with tumors, with a view to accumulating a sufficient number of instances of each disease to determine its characteristic course, the criteria for diagnosis, and to evaluate methods of treatment. To attain this objective, only those cases accompanied by comprehensive data and adequate material will be accepted for registration. While the chief purpose of the registry is the study of tumors, no limitation as to character of disease is contemplated. The cases preferred are those living at the time of registration and that can be followed so that the outcome may be ascertained. The following up of these cases will constitute a considerable and important part of the Registry's activities.

In some conditions, it is undoubtedly true that accurate diagnosis is not possible in the early stages when treatment offers the best chance of cure. It is only by the opportunity to see conditions early and then following through that interpretation of the changes and establishment of criteria for diagnosis can be learned at a time to be of benefit to the patient. In other cases there is less difficulty in diagnosis, but the methods of treatment need to be studied. For this purpose it is necessary to have a uniform classification of the conditions so that various methods

of treatment may really be comparable. This is particularly true in such conditions as tumors of the bladder, in which there are many types of tumor, all too frequently considered as similar.

Except at the large medical centers too few cases of any one kind can be accumulated to obtain results on which reasonable judgments can be based. Some of these centers have already established local registries. By combining the cases accumulated by the centers with the cases of the American Registry of Pathology obtained from the country as a whole, it will be possible to obtain considerable numbers in a much shorter time. It is the purpose of this registry to co-operate to the fullest extent with all such groups to the end that uniform nomenclature may be used and that the greatest amount of knowledge may be obtained in the shortest time.

The registry will never serve as a diagnostic laboratory replacing local pathologists. It is rather to be a clearing house in pathology to which will be sent cases already diagnosed, and the obscure cases about which more can be learned by obtaining the opinions of several pathologists. In addition, by sending "follow-up" letters to the physician registering cases, the Registry will be a means of helpful stimulation.

The foundation on which to build is now existent in the three registries already established: The Registry of Ophthalmic Pathology, of about 2,000 cases; the Registry of Lymphatic Tumors, of 200 cases; and the Registry of Bladder Tumors, of over 200 cases.

The collection and study of the cases in these registries has been done through the ophthalmological societies of this country, The American Association of Pathologists and Bacteriologists and the American Urological Association. The records so far collected have been obtained in spite of a minimum of publicity effort because there has been insufficient personnel available at the Army Medical Museum to conduct larger collections. As the registries have become better known, the rate of increase has risen rapidly, until at the present time the "follow-up" work alone is more than can be handled adequately in the time of the Museum personnel available for it. The expense of these registries has thus far been borne entirely by the Museum, whose entire budget, inclusive of all personnel and upkeep of plant, is about $30,000. The expense for materials and equipment for the proposed registry can be handled by the Museum, but it is necessary to have

additional help in the form of professional, clerical and technical personnel.

There is but one medical officer on duty at the Army Medical Museum. The Museum is the central pathological laboratory for the Army, and by official order, tissues and histories from all cases of tumor or suspected tumor are sent to the Museum from Army hospitals, which have a total bed capacity of 10,000. In addition protocols of all necropsies, some 800 per annum or about 50 per cent of all deaths at Army hospitals, are reviewed at the Museum, and tissues are examined from a considerable number of the necropsies. In all about 1500 of the 2000 annual accessions are from the military service, more than the personnel can handle satisfactorily. The clerical force at the Museum, one stenographer and one typist, cannot do more than the routine of the Army work. The drain on the technicians, of which there are two, is not great for the bladder tumors and lymphatic tumors, but the ophthalmic pathology requires over half of the time of one technician.

The fact that the officer personnel at the Museum is shifted every four years makes it necessary to provide a pathologist or registrar so that there will be assured a continuity of the policies outlined by the committee of the Registry. Even if the Army could allow sufficient personnel to handle the Registry, the War Department policy of requiring changes of station would result in uneven interest in, and development of, the Registry which requires active participation on the part of the registrar to make it of value. Well endowed research is now being conducted in many places on various phases of the tumor problem, and from this much good will come ultimately. In the meantime, however, there are many persons suffering from malignant disease who cannot be benefited by these studies but who afford a fertile field for investigation, promising quicker results in diminishing the fatality rate and prolonging life. The value of such investigations has been repeatedly demonstrated, but never better than by the Registry of bone tumors of the American College of Surgeons. By means of the study of these cases many lives have been saved and many deforming operations have been avoided. The Registry has made significant advances in our knowledge of the criteria for diagnosis, the outcome, course and methods of treatment of the bone tumors, and is still adding to our knowledge of these tumors.

In the present state of our understanding of neoplastic diseases more information can be obtained at less cost by registries than by any other manner. The information is immediately available to medical practice.

It is generally acknowledged by pathologists that many neoplasms are difficult to diagnose. In many the diagnosis is but a guess. The earliest changes which signify malignancy are not sharply defined. In fact, there is serious doubt if we know by sight the earliest malignant changes in any tissue. Unless cases are followed up we cannot learn these changes. Even if a definite cause for cancer should be found, it will still be necessary to recognize the earliest change indicating its presence.

Physicians are not justified in omitting the follow up of their cases, yet this is the rule and not the exception. Pressure of economic conditions and lack of facilities for adequate study are important factors. The proposed Registry will facilitate the "follow up" by the doctor by periodically sending to him a simple blank form on which he can make his report.

The Army Medical Museum is well equipped and has funds adequate for routine supplies. It is located in the same building as the Army Medical Library so that the literature is at hand for study. The proposed project of a new building to house both the Museum and Library at the Army Medical Center, adjacent to Walter Reed Hospital, is before the Bureau of the Budget. In the new building, as contemplated, there will be rooms available for research by scientists not on the Museum staff. Laboratory facilities will be available and all collections of the Museum will be more accessible for research. In this connection it is to be noted that an Act of Congress has already made available the collections of the Army Medical Museum to scientific men for research.

The registries as at present conducted furnish the Museum with duplicate records, and where possible duplicate slides from the cases to minimize the loss during the circulation of the material to the diagnostic committees.

The placing of this Registry under the auspices of the National Research Council insures continuity of administration and policy independent of Government action.

Funds donated to the Registry will be received and administered through the National Research Council by the National Academy of Sciences.

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*Attended 1930 meeting. Numbers in parentheses indicate years in which the Fellow became a member of the Academy. Fellows are requested to supply missing data and report errors in addresses or specialties to the Executive Secretary, Dr. W. P. Wherry, Medical Arts Bldg., Omaha, Nebr.

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*BUTLER, ROBERT H. (1920)
112 W. Columbus Ave., Bellefontaine, O., OALR
BUTT, MIRIAM M.....2045 Chestnut St., Philadelphia, Pa., Op.
BUVINGER, CHARLES W. (1921)
50 Washington St., East Orange, N. J., OALR
*BYRNES, H. F.....67 Chestnut St., Springfield, Mass., OALR

C

CADY, D. W. (1930).....65 N. Madison Ave., Pasadena, Cal., Op.
*CAHILL, J. W.....390 Main St., Worcester, Mass., OALR
CALDWELL, ROBERT
831 New Donaghey Bldg., Little Rock, Ark., OALR
CALHOUN, F. PHINIZY (1911)
486 Peach Tree St., N. E., Atlanta, Ga., Op.
CALHOUN, JAS. G.....Metropolitan Bldg., St. Louis, Mo., Op.
*CALLFAS, W. F.....1620 Med. Arts Bldg., Omaha, Neb., ALR
CAMERON, WM. G...Rhodes Med. Arts Bldg., Tacoma, Wash., OALR
*CAMP, W. E. (1920), Medical Arts Bldg., Minneapolis, Minn., OALR
*CAMPBELL, A. A. (1930).....86 Bloor St. W., Toronto, Ont., ALR
*CAMPBELL, C. H., Columbus State Bk. Bldg., Columbus, Neb., OALR
CAMPBELL, D. A.....David Whitney Bldg., Detroit, Mich., OALR
CAMPBELL, DON M. (1908)
David Whitney Bldg., Detroit, Mich., OALR
CAMPBELL, M. D. (1927)
David Whitney Bldg., Detroit, Mich., OALR
CAMPBELL, ROBERT A., 520 LaSalle Bldg., Minneapolis, Minn., OALR
CAMPBELL, W. K.....96 Third Ave., Long Branch, N. J., OALR
CAPRON, F. P.....118 Angell St., Providence, R. I., OALR
CARDWELL, EDGAR P. (1929).....15 Fulton St., Newark, N. J., ALR
CARLETON, RALPH (1914)....10 Temple St., Springfield, Mass., Op.
*CARMACK, J. W. (1919)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
*CARMODY, T. E. (1904).....Metropolitan Bldg., Denver, Colo., ALR
*CARMODY, THOMAS J. (1926)
408 The Temple Bldg., Danville, Ill., OALR

CARNCROSS, HELEN (1920)
58 E. Washington St., Chicago, Ill., Op.
CARPENTER, CHAPIN (1930)
1930 Chestnut St., Philadelphia, Pa., Op.
CARPENTER, E. R.....Medical Arts Bldg., Dallas, Tex., OALR
*CARPENTER, E. W. (1920).....Prof. Bldg., Greenville, S. C., OALR
CARR, GEO. W. (1909)....54 S. Franklin St., Wilkes-Barre, Pa., OALR
CARROLL, GEO. G.....5 N. Goodman St., Rochester, N. Y., OALR
CARRUTH, HOWARD ERNEST (1928)
Medical Arts Bldg., Portland, Ore., OALR
*CARSON, W. E.....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
*CARTER, E. D. (1930).....Central Bldg., Wichita, Kan., ALR
*CARTER, JOHN M. (1929)
David Whitney Bldg., Detroit, Mich., ALR
CARTER, W. W. (1896), 2 E. 54th St., New York City, N. Y., ALR
CARVILL, MAUD (1914)
82 Commonwealth Ave., Boston, Mass., Op.
CARY, ED. F. (1930)...171 W. 57th St., New York City, N. Y., ALR
*CARY, E. H.....Medical Arts Bldg., Dallas, Texas, OALR
CARY, G. C.....Canon Block, Grand Junction, Colo., OALR
*CASSADY, J. VERNAL (1928)
654 Associate Bldg., South Bend, Ind., OALR
*CAVANAUGH, J. A....1712-30 N. Michigan Blvd., Chicago, Ill., ALR
CAVE, LINUS S. (1929).....Physicians' Bldg., Syracuse, N. Y., ALR
*CHADWICK, IRA B. (1920).....715 Walnut, Coffeyville, Kan., Op.
CHANDLER, G. E. (1930).....Huntington Bldg., Miami, Fla., ALR
CHAPMAN, S. JEFFERSON (1926)
402 Burns Bldg., Colorado Springs, Colo., ALR
CHAPMAN, V. A.....110 E. Wisconsin Ave., Milwaukee, Wis., OALR
CHAPPELL, R. S. (1925)
Traction Terminal Bldg., Indianapolis, Ind., ALR
*CHARLES, JOS. W.....3720 Washington Ave., St. Louis, Mo., Op.
*CHARLTON, C. COULTER
124 S. Illinois Ave., Atlantic City, N. J., ALR
*CHASE, E. FRANK (1918).....1008 Cobb Bldg., Seattle, Wash., OALR
CHASE, S. B. (1925).....Carver Bldg., Ft. Dodge, Ia., OALR
CHASE, W. D. (1924).....230 E. Broad St., Bethlehem, Pa., ALR
CHATTIN, J. FRANKLIN.....671 Broad St., Newark, N. J., Op.
CHENERY, W. E.....377 Commonwealth Ave., Boston, Mass., ALR
CHENEY, JAMES W.....702 Schweiter Bldg., Wichita, Kan., OALR
CHILDERS, ROBT. J. (1929).....604 Park Ave., Plainfield, N. J., ALR
*CHILDS, H. A.....Eagle Blk., Creston, Ia., ALR
CHIPMAN, L. D. V. (1925)
42 Coburg St., St. John, N. B., Canada, OALR
*CHRISTOPH, CARL H. (1928)
30 N. Michigan Ave., Chicago, Ill., OALR
CLARK, H. S. (1916), Medical Arts Bldg., Minneapolis, Minn., OALR
*CLARK, I. G. (1920).....188 E. State St., Columbus, O., OALR
*CLARK, J. SHELDON (1914), 509 State Bk. Bldg., Freeport, Ill., OALR

*Clark, L. H. (1915).....337 Monroe Ave., Rochester, N. Y., OALR
Clay, G. E. (1917).....Medical Arts Bldg., Atlanta, Ga., Op.
*Cleff, Oscar (1926).....55 E. Washington St., Chicago, Ill., OALR
*Clement, C. C.....30 N. Michigan Blvd., Chicago, Ill., OALR
Clement, L. O. (1930).....Guardian Bldg., Salem, Ore., OALR
Clerf, Louis Henry (1923), 128 S. 10th St., Philadelphia, Pa., ALR
*Clevenger, W. F. (1905)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
Clifford, T. P. (1927)
1802 David Whitney Bldg., Detroit, Mich., OALR
Clothier, Joseph.....Kane Bldg., Pocatello, Idaho, OALR
*Coates, Geo. M. (1922).....1721 Pine St., Philadelphia, Pa., ALR
Cobb, Edwin (1912)
Masonic Temple Bldg., Marshalltown, Ia., OALR
Codd, A. N. (1929)
Paulsen Medical and Dental Bldg., Spokane, Wash., ALR
*Cody, Claude Carr, Jr. (1928)
1304 Walker Ave., Houston, Tex., ALR
Coffelt, Theodore A., 1240 Benton Ave., Springfield, Mo., OALR
Cogan, J. E.....707 Rose Bldg., Cleveland, O., OALR
Cohen, Herman B. (1927)
"The Lenox," Spruce and 13th Sts., Philadelphia, Pa., ALR
Cohen, Martin.....1 West 85th St., New York City, N. Y., Op.
Cohen, Samuel (1920).....2009 Pine St., Philadelphia, Pa., ALR
Cohn, Felix.....47 W. 87th St., New York City, N. Y., ALR
Cohn, Seldon.....Fulton, Ky., OALR
*Cohoe, Don A. (1930)
13535 Woodward Ave., Highland Park, Detroit, Mich., ALR
Colbert, R. M. (1929).... 57 West 57th St., New York, N. Y., ALR
*Collier, George B. (1929)
1317 Whitney Bldg., New Orleans, La., ALR
Collins, E. W.....508 Majestic Bldg., Denver, Colo., ALR
Collins, J. D. (1914).....187 Main St., Northampton, Mass., ALR
Colver, B. N. (1913).....Sanitarium 1, Battle Creek, Mich., ALR
*Comee, W. C. (1922).....Northern Bldg., Green Bay, Wis., OALR
Cone, Alfred J. (1929).....Laurel Bldg., Muscatine, Iowa, ALR
Conlon, F. A.....301 Essex St., Lawrence, Mass., OALR
*Connor, Chas. E. (1920).....Miller Clinic, St. Paul, Minn., ALR
*Connor, Ray (1905).....1410 Stroh Bldg., Detroit, Mich., OALR
Constans, George M. (1929)
Quain and Ramstad Clinic, Bismarck, N. D., Op.
Constantinople, P. S. (1929)
1801 Eye St., N. W., Washington, D. C., ALR
Conway, J. M. (1930).....Jenkins Arcade, Pittsburgh, Pa., ALR
*Cook, W. Albert (1924)....Medical Arts Bldg., Tulsa, Okla., OALR
Cooper, C. E.....652 Metropolitan Bldg., Denver, Colo., ALR
Copps, Lyman Alden (1923)
Marshfield Clinic, Marshfield, Wis., OALR

CORBETT, J. J. (1925).....520 Beacon St., Boston, Mass., OALR
*CORSER, J. B.....345 Wyoming Ave., Scranton, Pa., Op.
*CORTOPASSI, A. J. (1930)
Second Nat'l Bank Bldg., Saginaw, Mich., ALR
CORYA, H. W. (1930).....30 E. 40th St., New York, N. Y., ALR
COSTEN, J. B. (1925), Beaumont Medical Bldg., St. Louis, Mo., ALR
COTT, C. C.....104 Dorchester Road, Buffalo, N. Y., ALR
COTT, D. N. (1924)
25 Central Ave., St. George, Staten Isl., New York, ALR
COULTER, CHAS. F.
404 First Nat'l Bank Bldg., Great Falls, Mont., OALR
COWAN, ALFRED (1928), 1930 Chestnut St., Philadelphia, Pa., Op.
*COWLEY, ROBERT HENRY (1926).....Berea, Ky., OALR
*COWPER, H. W.....543 Franklin St., Buffalo, N. Y., Op.
CRAFT, KENNETH L. (1920)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
CRANE, J. WELLINGTON (1921)
128 Third Ave., Trenton, N. J., OALR
CRANE, W. R. (1929).....756 S. Broadway, Los Angeles, Cal., ALR
CREIGHTON, WM. J. (1910), 258 S. 18th St., Philadelphia, Pa., Op.
*CRISP, WM. H. (1913).....Metropolitan Bldg., Denver, Colo., Op.
CROCKETT, R. L.....302 Main St., Oneida, N. Y., Op.
CROMAN, JOS. M., JR. (1924)
115 Cass Ave., Mount Clemens, Mich., OALR
*CROSKEY, J. W. (1905).....Med. Arts Bldg., Philadelphia, Pa., Op.
*CROSS, A. E. (1914).....390 Main St., Worcester, Mass., Op.
CROSS, G. H. (1919).....525 Welsh St., Chester, Pa., Op.
*CROSSLEY, E. R.....30 N. Michigan Blvd., Chicago, Ill., OALR
*CRUMRINE, NORMAN ROSS (1928), 595 Third St., Beaver, Pa., OALR
CULBERTSON, L. R. (1898).....Masonic Temple, Zanesville, O., OALR
*CULLOM, M. M. (1910)
1119 Bennie-Dillon Bldg., Nashville, Tenn., OALR
CUMMINGS, GEO. O. (1930).....47 Deering St., Portland, Me., ALR
CUNNING, DANIEL S. (1928)
145 E. 54th St., New York City, N. Y., OALR
CUNNINGHAM, ALLAN R. (1924)
260 Barrington St., Halifax, N. S., OALR
CURDY, R. J.....609 Grand Ave., Kansas City, Mo., Op.
CURRAN, E. J. (1919)
1805 Federal Reserve Bk. Bldg., Kansas City, Mo., Op.
CURRY, GLENDON E. (1912)
Westinghouse Bldg., Pittsburgh, Pa., Op.
CURTIN, EUGENE A. (1923), 703 Connell Bldg., Scranton, Pa., OALR
CURTIS, W. L.....702 Sharp Bldg., Lincoln, Neb., OALR
*CUSHMAN, BEULAH (1924)
25 E. Washington St., Chicago, Ill., Op.
CUTINO, R. M. (1930).....165 Clinton St., Brooklyn, N. Y., Op.
CUTLER, FRANKLIN E.....R. D. No. 2, Cortland, N. Y., ALR

D

DABNEY, S. G.....Starks Bldg., Louisville, Ky., OALR
DABNEY, VIRGINIUS (1913)
1633 Connecticut Ave., Washington, D. C., ALR
DAILEY, GILBERT L.....618 N. 3rd St., Harrisburg, Pa., OALR
DAILY, LOUIS (1926).....1117 Med. Arts Bldg., Houston, Tex., OALR
*DAILY, RAY K. (1926), 1117 Med. Arts Bldg., Houston, Tex., OALR
DANCY, ALEXANDER B.
First Nat'l Bank Bldg., Jackson, Tenn., OALR
DANIELS, R. L.....48 E. Front St., New Bern, N. C., OALR
*DARMER, GEO. A. (1921).....401 Terminal Bldg., Aurora, Ill., OALR
*DARROW, C. H. (1928), 110 Metropolitan Bldg., Denver, Colo., ALR
*DAVIDSON, H. P. (1924)..58 E. Washington St., Chicago, Ill., Op.
DAVIDSON, MORRIS (1929), 210 W. 70th St., New York, N. Y., Op.
DAVIES, H. F. (1930), 1835 Eye St., N. W., Washington, D. C., ALR
DAVIES, J. S. (1927).....Washington Bldg., Tacoma, Wash., OALR
DAVIES, T. A. (1913).....578 Sherborne St., Toronto, Can., OALR
DAVIS, A. EDWARD.....40 E. 61st St., New York City, N. Y., Op.
DAVIS, CHARLES L. (1928)
3720 Washington Blvd., St. Louis, Mo., ALR
*DAVIS, F. A. (1927), 224 W. Washington Ave., Madison, Wis., OALR
DAVIS, G. E.....1060 Fifth Ave., New York City, N. Y., OALR
*DAVIS, HARRY B. (1920), Waldheim Bldg., Kansas City, Mo., OALR
DAVIS, J. CALVIN (1928).....425 Aquila Court, Omaha, Neb., OALR
DAVIS, J. E.....Kress Bldg., McAlester, Okla., OALR
DAVIS, J. L.....135 S. 18th St., Philadelphia, Pa., ALR
DAVIS, RALPH F. (1920).....Mayer Bldg., Portland, Ore., OALR
*DAVIS, THOMAS W.
210 O'Hanlon Bldg., Winston-Salem, N. C., OALR
*DAVIS, WM. C. (1930).....327 E. State St., Columbus, O., Op.
*DAVIS, W. T. (1919).....927 Farragut Sq., Washington, D. C., Op.
DAY, KENNETH M. (1930)
121 University Place, Pittsburgh, Pa., ALR
DAYTON, G. O.....1401 S. Hope St., Los Angeles, Cal., OALR
DEAL, JOHN FRANCIS (1928)
First Nat'l Bank Bldg., Springfield, Ill. OALR
*DEAN, ALFRED, 308 Medical Arts Bldg., Grand Rapids, Mich., Op.
*DEAN, ARTHUR C. (1929), 115 River Ave., Hot Springs, S. D., ALR
DEAN, F. W. (1904).....532 First Ave., Council Bluffs, Ia., OALR
*DEAN, L. W.
Medical School, Washington University, St. Louis, Mo., ALR
DEAN, WALTER (1917).....300 Francis Bldg., Louisville, Ky., OALR
DEBOE, M. P.....1st Nat'l Bank Bldg., Miami, Fla., OALR
DECHERD, H. B.....1515 Medical Arts Bldg., Dallas, Tex., OALR
DECHTER, M. ARCHIBALD (1930)
Professional Bldg., Westfield, Mass., ALR
*DECKER, J. C. (1918).....Frances Bldg., Sioux City, Ia., OALR

*DEFNET, WM. A. (1921)..28 Adams Ave., West Detroit, Mich., ALR
DEICHLER, LYN W.....1737 Chestnut St., Philadelphia, Pa., OALR
*DELPH, JOHN FRANCIS (1928)
104 S. Michigan Blvd., Chicago, Ill., ALR
DENMAN, IRA O. (1915).....330 Richardson Bldg., Toledo, O., OALR
DENNIS, DAVID N. (1902).....221 W. Ninth St., Erie, Pa., Op.
DENNIS, EDWARD P. (1921).....309 W. 10th St., Erie, Pa., OALR
DENNIS, F. L. (1906)
301 Ferguson Bldg., Colorado Springs, Colo., ALR
*DENNIS, GEO. J. (1910)....25 E. Washington St., Chicago, Ill., ALR
DEPPING, C. W. (1930).....17 W. 54th St., New York, N. Y., ALR
DERBY, GEO. S.....5 Bay State Rd., Boston, Mass., Op.
DERIVER, J. P. (1919), Veterans Bureau, San Francisco, Cal., OALR
DETLING, FRANK E.....500 S. Lucas Ave., Los Angeles, Cal., OALR
DEWEESE, CLARENCE....201 West Second St., Lexington, Ky., OALR
*DEWEY, C. H. (1912), Maj. M. C., U. S. Army,
U. S. Army Hosp., Ft. Benj. Harrison, Indianapolis, Ind., OALR
DEWITT, EDWARD NICHOLAS (1928)
836 Myrtle Ave., Bridgeport, Conn., Op.A
*DICKSON, JOHN FORREST (1928)
418 Mayer Bldg., Portland, Ore., OALR
*DILLON, IRA H. (1930)....Hatcher Hospital, Wellington, Kan., Op.
DINTENFASS, HENRY.....1305 Spruce St., Philadelphia, Pa., ALR
*DIXON, FRED W. (1929).....Rose Bldg., Cleveland, O., ALR
DIXON, G. S.....1150 5th Ave., New York City, N. Y., R
DIXON, OTTO JASON (1928)
902 Medical Arts Bldg., Kansas City, Mo., ALR
DIXON, W. E., 503 Medical Arts Bldg., Oklahoma City, Okla., OALR
DLUGASCH, LOUIS (1929), 110 E. 36th St., New York, N. Y., ALR
DOANE, L. L. (1905).....357 N. Main St., Butler, Pa., OALR
*DOBBS, O. R. (1930).....2435 N. Grand Blvd., St. Louis, Mo., ALR
DODD, OSCAR (1903).....636 Church St., Evanston, Ill., Op.
*DOHERTY, WM. B. (1927)
150 W. 55th St., New York City, N. Y., Op.
DOLMAN, PERCIVAL (1919), 490 Post St., San Francisco, Cal., Op.
DONNELL, N. R. (1917)....Metropolitan Bldg., St. Louis, Mo., Op.
DONOHER, WM. D.....Boston Bldg., Salt Lake City, Utah, OALR
DONOVAN, JNO. A. (1902)....507 Phoenix Bldg., Butte, Mont., OALR
*DORENTE, D. R.....1st Nat'l Bank Bldg., Fort Smith, Ark., OALR
DORGE, RICHARD IRVING (1922)
2319 Sixth St., South, Minneapolis, Minn., OALR
*DOUGLAS, FREDERICK ALEXANDER (1922)
303 State Bank Bldg., La Crosse, Wis., OALR
DOW, FRANK E. (1914).....4 West St., Northampton, Mass., Op.
DOWLING, J. IVIMEY.....116 Washington Ave., Albany, N. Y., OALR
DOWLING, J. T. (1915).....1115 Terry, Seattle, Wash., OALR
*DUANE, J. F. (1916).....Jefferson Bldg., Peoria, Ill., OALR
DULANEY, OCTAVUS.....811 Brown Bldg., Louisville, Ky., ALR
DUNCAN, R. A. (1929)..339 Amarillo Bldg., Amarillo, Texas, ALR

DUNLAP, LAWRENCE G. (1920)
101 Main St., Anaconda, Mont., OALR
*DUNN, MURISON ..... Richmond, Ky., OALR
DUNN, O. B. ..... 4th and Railroad Sts., Ironton, O., OALR
*DUNNINGTON, J. H. (1924)
30 W. 59th St., New York City, N. Y., Op.
*DUNTLEY, G. S. (1913) ..... 103 So. Side Sq., Macomb, Ill., OALR
DURR, S. A. (1927)
1304 Medico-Dental Bldg., San Diego, Cal., Op.
*DUTROW, H. V. (1914) ..... 1040 Fidelity Med. Bldg., Dayton, O., OALR
DYER, B. G. (1929) ..... Ames, Iowa, ALR
DYER, C. P. (1920) ..... 408 Humboldt Bldg., St. Louis, Mo., Op.
DYER, WALLACE C. (1929)
Central Union Bank Bldg., Evansville, Ind., ALR

E

*EAGLETON, W. P. (1930) ..... 15 Lombardy St., Newark, N. J., ALR
*EAREL, A. M. (1912) ..... Willdon Bldg., Hoopeston, Ill., OALR
EARLEY, JAMES HUGH (1928)
1801 Eye St., N. W., Washington, D. C., ALR
EASTON, J. C. (1904), First Nat'l Bank Bldg., Springfield, O., OALR
EBELING, WM. B. (1930) ..... 54 Greene Ave., Brooklyn, N. Y., Op.
EBER, CARL THEODORE (1928)
1006 Carleton Bldg., St. Louis, Mo., Op.
EBERT, EDWIN C. (1929)
Naval Dispensary, Washington, D. C., ALR
EDSON, R. A. ..... 498 Delaware Ave., Buffalo, N. Y., Op.
EDWARDS, C. J. (1913)
First National Bank Bldg., Vicksburg, Miss., OALR
EDWARDS, SLOCOMB R. (1918)
Christman Bldg., South Bend, Ind., OALR
*EGAN, BURTON WRIGHT (1928)
408 North St., Logansport, Ind., OALR
*EIMER, CHARLES EUGENE (1928)
316 Lister Bldg., St. Louis, Mo., ALR
*ELLETT, E. C. (1898) ..... Exchange Bldg., Memphis, Tenn., Op.
*ELLIS, CHARLES WARD (1928)
215 N. Walnut St., Lansing, Mich., ALR
ELLIS, Z. H. (1927) ..... 58 E. 65th St., New York City, N. Y., Op.
*ELWOOD, CALVIN R. (1922) ..... Spies Bldg., Menominee, Mich., OALR
EMBERY, FRANK (1927)
1532 Overington St., Philadelphia, Pa., ALR
EMERSON, LINN (1906) ..... Metropolitan Bldg., Orange, N. J., OALR
ENGLE, H. P. ..... Maytag Bldg., Newton, Ia., OALR
ERSNER, MATTHEW S. ..... 1915 Spruce St., Philadelphia, Pa., ALR
ESTABROOK, CHAS. T. ..... 857 Pleasant St., Worcester, Mass., OALR

*Eubank, Ambrose E. (1928)
636 Argyle Bldg., Kansas City, Mo., OALR
*Evans, J. N. (1925)....23 Schermerhorn St., Brooklyn, N. Y., Op.
Ezell, H. C. (1926).....228 Doctors Bldg., Nashville, Tenn., OALR

F

Fackler, C. L. (1930).....11 E. Market St., York, Pa., ALR
Fairbairn, John F.....925 Delaware Ave., Buffalo, N. Y., ALR
*Fairchild, Nora M. (1922)...Med. Arts Bldg., Omaha, Neb., Op.
Fairing, John W. (1924)
206 Tribune Review Bldg., Greensburg, Pa., OALR
*Faith, Thomas.....31 N. State St., Chicago, Ill., OALR
*Fallows, Howard D. (1916)
Park Hospital Clinic, Mason City, Ia., OALR
*Farmer, A. G. (1919).....Fidelity Medical Bldg., Dayton, O., OALR
*Farnsler, H. Hershey.....1438 Market St., Harrisburg, Pa., OALR
Farnsworth, M. A. (1917)
Chamberlain Sanitarium and Hospital, Chamberlain, S. D., OALR
Farrell, P. J. H. (1902), 25 E. Washington St., Chicago, Ill., OALR
Farrington, P. M. (1904)
Physicians and Surgeons Bldg., Memphis, Tenn., ALR
Farrior, J. Brown (1928)
605 Citizens Bank Bldg., Tampa, Fla., OALR
*Fawcett, I. (1913).....75 12th St., Wheeling, W. Va., OALR
Felt, C. L.....Chestnut and 20th Sts., Philadelphia, Pa., ALR
*Fenton, Ralph A. (1921)
806 Medical Arts Bldg., Portland, Ore., OALR
*Ferguson, E. S. (1906)
902 Medical Arts Bldg., Oklahoma City, Okla., OALR
Ferguson, R. J. (1927)....59 College St., New Haven, Conn., OALR
Ferman, John W. (1921).....Stahlman Bldg., Charleroi, Pa., OALR
Fernlund, George Byron (1928)
Hillside Ave. and 118th St., Richmond Hill, New York, ALR
Ferris, David P. (1928), 1000 Carleton Bldg., St. Louis, Mo., ALR
Fielding, F. G. (1925).....70 Ridge St., Glen Falls, N. Y., ALR
*Figi, F. A. (1927).....Mayo Clinic, Rochester, Minn., LR
Findlay, E. K. (1930).....30 N. Michigan Ave., Chicago, Ill., Op.
Fineberg, Maxwell (1930)
3858 Westminster Place, St. Louis, Mo., ALR
*Fink, Walter H. (1927)
Medical Arts Bldg., Minneapolis, Minn., OALR
*Finnoff, W. C. (1916).....Republic Bldg., Denver, Colo., Op.
Fischer, N. A. (1925), 623 Union Trust Bldg., Pittsburgh, Pa., ALR
Fischer, V. B.....203 Mercantile Bk. Bldg., Boulder, Colo., OALR
Fisher, Carl.....1930 Wilshire Blvd., Los Angeles, Cal., Op.
Fisher, James A. (1925)
Jersey Central Bldg., Asbury Park, N. J., OALR

*Fisher, W. A. (1896), 231 W. Washington St., Chicago, Ill., OALR
Fiske, Geo. F.....25 E. Washington St., Chicago, Ill., OALR
*Fitzgerald, J. P. (1924), 30 N. Michigan Blvd., Chicago, Ill., Op.
Flagg, J. D. (1900).....158 Anderson Place, Buffalo, N. Y., Op.A.
*Flatley, T. J. (1914).....1314 5th Ave., Moline, Ill., OALR
Floyd, T. W. (1918).....811 Peoria Life Bldg., Peoria, Ill., OALR
Flynn, Chas. T. (1927)
41 Trumbull St., New Haven, Conn., ALR
Flynn, James A. (1924)
1511 Rhode Island Ave., N. W., Washington, D. C., OALR
*Fogarty, Chas. W. (1920)
St. Paul Clinic Bldg., St. Peter and 5th St., St. Paul, Minn., OALR
Foley, J. W. (1915).....Majestic Bldg., Denver, Colo., OALR
Folk, Max Lyon (1928), 55 E. Washington St., Chicago, Ill., OALR
Folken, Frank Garrett (1928)
306 Albert Lea State Bank Bldg., Albert Lea, Minn., OALR
*Folsom, W. H. (1927).....11 N. Main St., Fond du Lac, Wis., OALR
Forbes, Sherman B. (1921)
409 Citizens Bank Bldg., Tampa, Fla., OALR
*Ford, Hanby Lewis (1928)
401 Robeson Bldg., Champaign, Ill., OALR
*Forgrave, L. Robert (1922), 204 Logan Bldg., St. Joseph, Mo., OALR
*Forsythe, S. T. (1919).....Rose Bldg., Cleveland, O., OALR
*Fort, A. G.....303 Medical Arts Bldg., Atlanta, Ga., OALR
Foster, J. H.....1304 Walker Ave., Houston, Tex., ALR
*Fouts, John D. (1920).....910 Fidelity Bldg., Dayton, O., OALR
*Fowler, Earl B. (1925)..55 E. Washington St., Chicago, Ill., Op.
Fowler, Robt. H. (1929)..153 E. 62nd St., New York, N. Y., ALR
Fowler, S. R. (1910).....University Blk., Syracuse, N. Y., ALR
Fowler, Wm. (1925).....1048 Maccabee Bldg., Detroit, Mich., OALR
Fowlkes, John W.....30 W. 59th St., New York, N. Y., ALR
Fox, C. H. (1920).....5 W. 21st St., Kearney, Neb., OALR
Fox, L. Webster.....303 S. 17th St., Philadelphia, Pa., Op.
*Fox, Noah (1926).....841 E. 63rd St., Chicago, Ill., ALR
*Fox, Ralph D. (1916).....Unity Bldg., Bloomington, Ill., OALR
Franchere, Fred'k E. (1920)
Toy Bank Bldg., Sioux City, Ia., OALR
*Frank, Ira (1908).....104 S. Michigan Blvd., Chicago, Ill., ALR
Frankel, Edward, Jr. (1928)
152 W. 58th St., New York City, N. Y., ALR
Fraser, R. H. (1925)
Battle Creek Sanitarium, Battle Creek, Mich., ALR
Fraser, Robt. C. (1929), 940 Military St., Port Huron, Mich., ALR
Fraser, W. G. (1927).....Medical Arts Bldg., Ottawa, Canada, OALR
Freedman, L. M. (1920)
475 Commonwealth Ave., Boston, Mass., ALR
*Freimuth, L. E. (1927)
University Club Bldg., St. Louis, Mo., ALR
*French, R. F. (1913).....Masonic Temple, Marshalltown, Ia., OALR

FRIEDENWALD, HARRY.....1212 Eutaw Pl., Baltimore, Md., Op.A.
*FRIEDENWALD, J. S. (1925)....1212 Eutaw Pl., Baltimore, Md., Op.
*FRINGER, W. R.....William Brown Bldg., Rockford, Ill., Op.
*FROST, ALBERT DALBEY (1928)
681 E. Broad St., Columbus, O., Op.
FROTHINGHAM, EDWARD (1928)
8644 94th St., Woodhaven, N. Y., OALR
FUCHS, VALENTINE HENRY (1928)
220 Chaille Bldg., New Orleans, La., ALR
FULKERSON, C. B. (1914)
308 Kalamazoo Nat'l Bank Bldg., Kalamazoo, Mich., OALR
FULLENWIDER, C. M.....Barnes Bldg., Muskogee, Okla., OALR
*FULLER, HARRY B. (1925)....2219 E. 70th Place, Chicago, Ill., OALR
FULLER, JOHN ANDRE (1928)
First National Bank Bldg., Reno, Nev., OALR
*FULLER, T. E. (1915), 100 West Broad St., Texarkana, Tex., OALR
*FULTON, JOHN F. (1920)..833 Lowry Bldg., St. Paul, Minn., OALR
FURGASON, A. P.....Richards Blk., Lincoln, Neb., OALR
*FURSTENBERG, A. C. (1925)
First National Bank Bldg., Ann Arbor, Mich., ALR
*FUST, JOHN H. (1922).....221 W. 9th St., Erie, Pa., OALR

G

GABRIEL, C. K. (1930)
Western Catholic Union Bldg., Quincy, Ill., ALR
GADBOIS, ARTHUR E. (1921), 421 Norfolk Ave., Norfolk, Neb., OALR
*GAILEY, WATSON W. (1923)
Griesheim Bldg., Bloomington, Ill., OALR
GALE, M. JEAN (1919).....Republic Bldg., Denver, Colo., Op.
GALLAGHER, J. H. C. (1927), 1537 Main St., Springfield, Mass., ALR
GALLAHER, T. J.....California Bldg., Denver, Colo., ALR
*GALLOWAY, T. C. (1927).....636 Church St., Evanston, Ill., ALR
*GAMBLE, R. C. (1929).....30 N. Michigan Ave., Chicago, Ill., Op.
*GARBER, J. M.....14 N. Mulberry St., Mansfield, O., OALR
*GARRAGHAN, EDW. F.....25 E. Washington St., Chicago, Ill., OALR
GARRETT, J. R. (1913).....Mt. Trust Bldg., Roanoke, Va., OALR
GATEWOOD, E. T. (1925), Professional Bldg., Richmond, Va., ALR
GATEWOOD, W. L.....330 Park Ave., New York City, N. Y., ALR
GAUDET, LUCIEN SIDNEY (1928)
405 Franklin St., Natchez, Miss., OALR
*GEIGER, A. H. (1929)....554 Fullerton Parkway, Chicago, Ill., ALR
*GEIGER, C. W. (1904).....Arcade Bldg., Kankakee, Ill., OALR
*GEORGE, HENRY W. (1919), 19 N. Union St., Middletown, Pa., Op.
*GIFFORD, S. R. (1919).....58 E. Washington St., Chicago, Ill., Op.
GILBERT, FRANK Y.....148 Park St., Portland, Me., OALR
GILL, ARTHUR ERNEST (1928)
U. S. Veterans' Hosp., Fort Bayard, N. M., OALR
GILL, E. G. (1919).....711 S. Jefferson St., Roanoke, Va., ALR

GILL, G. BACHE (1923)
1624 Eye St., N. W., Washington, D. C., ALR
GILL, GEORGE (1917)
Elyria Savings and Trust Bldg., Elyria, O., OALR
GILL, WM. DAVIS (1929)
323 Medical Arts Bldg., San Antonio, Texas, OALR
GILLETT, WILBUR G.....201½ N. Main St., Wichita, Kan., Op.
GILLETTE, D. F. (1925)....State Tower Bldg., Syracuse, N. Y., Op.
GILLILAND, O. S. (1930), Professional Bldg., Kansas City, Mo., ALR
*GILLUM, J. R. (1920).....221 S. 6th St., Terre Haute, Ind., OALR
GIPNER, J. F. (1926).....53 S. Fitzhugh St., Rochester, N. Y., Op.
*GITTENS, T. R. (1920)....401 Davidson Bldg., Sioux City, Ia., OALR
GLASFORD, S. T. (1924).....Farmers' Bank Bldg., Pekin, Ill., OALR
GLEASON, E. B.....2112 Pine St., Philadelphia, Pa., ALR
*GLICK, HARRY NAYLOR (1928)
3115 S. Grand Ave., St. Louis, Mo., ALR
*GLOSSER, HERBERT H. (1912)
448 Franklin St., Buffalo, N. Y., Op.
*GLOVER, S. P.....1118 Twelfth Ave., Altoona, Pa., OALR
GLOWACKI, BEN FRANCIS (1928)
770 Maccabee Bldg., Detroit, Mich., OALR
GOAR, EVERETT LOGAN (1926)
1300 Walker Ave., Houston, Tex., Op.
GODDARD, H. M.....1531 Spruce St., Philadelphia, Pa., ALR
GOLDBACK, L. J. (1911), 1012 N. Charles St., Baltimore, Md., Op.
GOLDING, JOSEPH E. (1925), 170 Hancock St., Brooklyn, N. Y., Op.
*GOLDMAN, A. MILTON (1928)
121 E. 60th St., New York, N. Y., Op.
GOLDMAN, H. G.....57 W. 58th St., New York City, N. Y., ALR
*GOLDSMITH, PERRY G.....Medical Arts Bldg., Toronto, Can., ALR
*GOLDSTEIN, ISADORE (1929), 1060 Park Ave., New York, N.Y., Op.
GOLDSTEIN, M. A. (1896), 3858 Westminster Pl., St. Louis, Mo., ALR
GOOD, WM. M. (1929).....63 Center St., Waterbury, Conn., ALR
GOODE, ED. A. (1929).....239 Sanford Ave., Flushing, N. Y., ALR
*GOODYEAR, H. M. (1924).....Doctors Bldg., Cincinnati, O., ALR
GORDON, CHAS. H. (1926).....46 Deering St., Portland, Me., ALR
GORDON, GLENN G. (1929)..1st Nat'l Bank Bldg., Baker, Ore., ALR
*GORDON, J. S. (1919)....141 Wisconsin Ave., Milwaukee, Wis., OALR
GORRELL, T. J. H.
16th and Vincennes Ave., Chicago Heights, Ill., OALR
GOSNEY, C. W. (1920), Medical Arts Bldg., Los Angeles, Cal., Op.
*GOTTLIEB, MARK J.....2 West 87th St., New York, N. Y., ALR
*GOURLEY, G. F. (1916).....Sinclair Bldg., Steubenville, O., OALR
*GOUTERMAN, J. I.....1607 W. Diamond St., Philadelphia, Pa., Op.
GOUX, RAYMOND SALOT
545 David Whitney Bldg., Detroit, Mich., OALR
GOWEN, FRANCIS A. (1929), 2033 Pine St., Philadelphia, Pa., ALR
GOWEN, GEO. R. (1929)
306 Baker Bldg., Walla Walla, Wash., ALR

*GRACEY, GEO. F.....209 State St., Harrisburg, Pa., OALR
*GRADLE, H. S. (1911).....58 E. Washington St., Chicago, Ill., Op.
GRAHAM, H. B.....490 Post St., San Francisco, Cal., ALR
GRANGER, W. B. (1920)
Citizens Nat'l Bank Bldg., Emporia, Kan., OALR
*GRANT, FRED E. (1912)
Kalamazoo Nat'l Bank Bldg., Kalamazoo, Mich., OALR
*GRANT, H. W. (1930).....Hamm Bldg., St. Paul, Minn., Op.
*GRANT, LEE E. (1919)..David Whitney Bldg., Detroit, Mich., OALR
*GRANT, P. T. (1919)
1407 Grand Rapids Nat'l Bk. Bldg., Grand Rapids, Mich., OALR
GRAY, E. B. (1927)
909 Montgomery Bldg., Spartansburg, S. C., ALR
*GREEAR, J. N. JR. (1929)
1740 M St., N. W., Washington, D. C., Op.
*GREEN, A. S.
The Green's Eye Hosp., 1801 Bush St., San Francisco, Cal., Op.
*GREEN, JOHN (1905), 3720 Washington Blvd., St. Louis, Mo., Op.
GREEN, L. D.
The Green's Eye Hosp., 1801 Bush St., San Francisco, Cal., Op.
GREENE, JOSEPH BERRY (1923)
305 Haywood Bldg., Asheville, N. C., ALR
GREENE, LAURENCE WHITRIDGE (1928)
Republic Bldg., Denver, Colo., ALR
GREENE, M. L. (1930), Missouri Theater Bldg., St. Louis, Mo., Op.
GREENFIELD, SAMUEL D. (1927)
169 New York Ave., Brooklyn, N. Y., ALR
GREENFIELD, WILLIAM JOHN (1928)
50 Anderson St., Hackensack, N. J., ALR
*GREENWOOD, ALLEN (1911)
82 Commonwealth Ave., Boston, Mass., Op.
*GREENWOOD, G. J. (1930).....317 1st Ave., East, Newton, Ia., ALR
GREEVER, B. L. (1920)
610 Rorabaugh-Wiley Bldg., Hutchinson, Kan., OALR
GREGG, JOHN B.
612 Security Nat'l Bank Bldg., Sioux Falls, S. D., OALR
GRESSER, ED. B. (1929).....39 Fifth Ave., New York, N. Y., Op.
*GRIFFEY, ED. W. (1929), 1300 Walker Ave., Houston, Texas, ALR
GRIFFITH, H. M. (1930)
Johnstown Trust Bldg., Johnstown, Pa., Op.
*GRIM, U. J.....30 N. Michigan Blvd., Chicago, Ill., OALR
GRISCOM, J. MILTON.....2213 Walnut St., Philadelphia, Pa., Op.
GROSS, JULIUS E. (1920)
8030 Jenkins Arcade Bldg., Pittsburgh, Pa., ALR
GROSVENOR, L. N. (1907), 366 Dakota Ave., So. Huron, S. D., OALR
*GROVE, W. E. (1914), 120 E. Wisconsin Ave., Milwaukee, Wis., OALR
GRUSHLOW, ISRAEL
271 Central Park W., New York City, N. Y., ALR
*GSELL, J. F. (1920).....911 Beacon Bldg., Wichita, Kan., OALR

GUERINOT, A. J. (1918)
604 Westinghouse Bldg., Pittsburgh, Pa., OALR
*GUGGENHEIM, L. K.....Carleton Bldg., St. Louis, Mo., ALR
*GUNDELACH, C. A., 825 University Club Bldg., St. Louis, Mo., ALR
GUNN, M. L. (1929).....Smith Bldg., Harlan, Ky., ALR
GUTHRIE, A. L. (1910)....Osler Bldg., Oklahoma City, Okla., OALR
GUTTMAN, J.....1235 Park Ave., New York City, N. Y., OALR
*GUTTMAN, M. R. (1929)....185 N. Wabash Ave., Chicago, Ill., ALR
*GUYTON, B. S. (1918)....512 Van Buren Ave., Oxford, Miss., OALR
GWINN, G. E. (1916)
704 Central Trust Bldg., San Antonio, Texas, OALR

H

*HAAS, FREDERICK J. (1920)
Masonic Bldg., Leavenworth, Kan., OALR
HACKNEY, F. J. (1913)
630 Volunteer Bldg., Chattanooga, Tenn., OALR
HADEN, HENRY C.....1914 Travis St., Houston, Texas, Op.
*HAESSLER, F. HERBERT (1929)
120 E. Wisconsin Ave., Milwaukee, Wis., Op.
HAGEMANN, JOHN A. (1925), Highland Bldg., Pittsburgh, Pa., ALR
HAGLER, E. E. (1896)....401 E. Capitol Ave., Springfield, Ill., OALR
HAHN, WM. H. (1930).....15 Lombardy St., Newark, N. J., Op.
HALE, ARTHUR STANLEY (1928)
1609 Eaton Tower, Detroit, Mich., OALR
*HALL, ALICE K. (1924).....25 E. Washington St., Chicago, Ill., ALR
HALL, EDWIN PERRY (1928)
State Tower Bldg., Syracuse, N. Y., ALR
*HALL, G. C. (1907).....705 Brown Bldg., Louisville, Ky., OALR
HALLETT, G. DEWAYNE (1927)
274 W. 86th St., New York City, N. Y., Op.
HALSTED, FRED S.....738 Metropolitan Bldg., Denver, Colo., OALR
HAMLIN, FRED EUGENE (1926)
Shenandoah Life Bldg., Roanoke, Va., ALR
*HAMPSEY, A. R. (1930).....May Bldg., Pittsburgh, Pa., ALR
HAMPTON, ROBT. R.....804 Boston Bldg., Salt Lake City, Utah, ALR
HANCOCK, J. C. (1914), 135 Cambridge Pl., Brooklyn, N. Y., Op.
Also 2 Rector St., New York, N. Y.
*HANDS, SIDNEY GILLOTT (1926)
201 West 2nd St., Davenport, Ia., ALR
*HANEY, WM. P. (1920)
1500 Medical Arts Bldg., Omaha, Neb., OALR
HANS, C. L. (1921).....Provident Bank Bldg., Cincinnati, O., OALR
*HANSEL, FRENCH KELLER (1926)
Beaumont Medical Bldg., St. Louis, Mo., ALR
*HANSEN, ERLING W. (1920)
Medical Arts Bldg., Minneapolis, Minn., OALR

HANSON, WM. L. (1929)
318 Murphy Bldg., East St. Louis, Ill., ALR
*HARALSON, CHAS. H. (1929)
816 Medical Arts Bldg., Tulsa, Okla., Op.
HARBERT, JOHN P. (1918)..135 N. Main St., Bellefontaine, O., OALR
HARDESTY, J. F. (1920).....Missouri Bldg., St. Louis, Mo., Op.
HARDIE, G. C. (1930)....290 W. Michigan Ave., Jackson, Miss., ALR
*HARDY, LE GRAND HAVEN (1928)
30 E. 40th St., New York City, N. Y., Op.
*HARDY, W. F. (1914).....Humboldt Bldg., St. Louis, Mo., Op.
*HARGITT, CHARLES A. (1926)
17 Schermerhorn St., Brooklyn, N. Y., Op.
*HARKNESS, GORDON F. (1911), Putnam Bldg., Davenport, Ia., OALR
*HARRELL, VOSS (1926).....Hofman Bldg., Detroit, Mich., ALR
HARRIS, C. M. (1910)
604 Johnstown Trust Bldg., Johnstown, Pa., OALR
HARRIS, ED. W. (1930).....681 E. Broad St., Columbus, O., ALR
HARRIS, H. B.....Fidelity Med. Bldg., Dayton, O., OALR
HARRIS, THOMAS J. (1929), 104 E. 40th St., New York, N. Y., ALR
HARRIS, W. C. (1900).....Groton Bldg., Cincinnati, O., ALR
HARRIS, WM. ROBIN (1923)
500 Lamar Life Bldg., Jackson, Miss., ALR
*HARRISON, FRED HAZARD (1928)
308 First Nat'l Bank Bldg., Connellsville, Pa., OALR
HARRISON, W. G.....Empire Bldg., Birmingham, Ala., OALR
HARTER, JOHN H. (1929)....320 Stimson Bldg., Seattle, Wash., ALR
HARTLEY, F. A.....302 E. High St., Springfield, O., OALR
HARTSHORNE, ISAAC (1926)
30 W. 59th St., New York City, N. Y., Op.
HARTZELL, S. M.
Dollar Savings and Trust Co., Youngstown, O., OALR
*HARVEY, N. D.....112 Waterman St., Providence, R. I., OALR
*HASELTINE, BURTON (1927)
122 S. Michigan Ave., Chicago, Ill., OALR
HASKELL, ALFRED W.....129 Park St., Portland, Me., Op.
HASKIN, W. H. (1913).....57 W. 57th St., New York, N. Y., ALR
HASPEL, M. DAVID (1911)
603 Maison Blanche Bldg., New Orleans, La., OALR
HASTY, F. E. (1925).....Medical Arts Bldg., Nashville, Tenn., ALR
HATCH, C. B. (1908).....3 W. Church St., Newark, O., OALR
*HAUER, A. M. (1916).....327 E. State St., Columbus, O., ALR
HAUGHEY, WILFRED H. (1911)
303 Post Bldg., Battle Creek, Mich., OALR
HAWLEY, C. W.....Medical Arts Bldg., Chicago, Ill., OALR
HAWMAN, ERLE G.....4051 N. Broad St., Philadelphia, Pa., Op.
*HAYDEN, AUSTIN A. (1910)
25 E. Washington St., Chicago, Ill., OALR
HAYS, HAROLD M. (1912)
64 E. 58th St., New York City, N. Y., ALR

*Hazlett, L. R. (1913), Savings and Trust Bldg., Butler, Pa., OALR
Healey, John F. (1924)..503 Delaware Ave., Buffalo, N. Y., ALR
*Heard, C. F. (1913).....813 Sassafras St., Erie, Pa., OALR
Heard, Mary K.....128 19th Ave., North, St. Petersburg, Fla., Op.
*Heath, Parker (1924), David Whitney Bldg., Detroit, Mich., Op.
Hebert, Arthur W.....450 Sutter St., San Francisco, Cal., OALR
*Heckel, Edward B. (1902)
Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
Hedrick, Harold B. (1929)
910 Rialto Bldg., Kansas City, Mo., ALR
Heeb, Harry J.....204 Wisconsin Ave., Milwaukee, Wis., OALR
Heed, C. R.....1205 Spruce St., Philadelphia, Pa., Op.
Heetderks, D. R. (1930)
Medical Arts. Bldg., Grand Rapids, Mich., ALR
Heggie, N. M. (1905).....Buckman Bldg., Jacksonville, Fla., OALR
*Heitger, J. D. (1905).....First Trust Bldg., Pasadena, Cal., OALR
Helfrich, Ed. D. (1930).....269 E. State St., Columbus, O., ALR
Hempstead, Bert E.....Mayo Clinic, Rochester, Minn., ALR
*Henderson, L. E. (1930), 168 Sterling St., Watertown, N. Y., Op.
Hennessey, Wm. W.....333 Essex St., Salem, Mass., OALR
Henning, Carl.....1835 Eye St., N. W., Washington, D. C., Op.
Henninger, L. L. (1913)..Professional Bldg., Pasadena, Cal., OALR
Henton, G. E. (1918).....Morgan Bldg., Portland, Ore., OALR
Herriman, L. L. (1913).....Alamosa, Colo., OALR
Hess, Wm. L. (1904).....7478 Eads Ave., La Jolla, Cal., OALR
Hetrick, L. E. (1923), 40 E. 61st St., New York City, N. Y., ALR
Hetzel, C. C. (1906).....Eccles Bldg., Ogden, Utah, OALR
*Hickey, H. L. (1930).....Republic Bldg., Denver, Colo., ALR
Hicks, Vonnie Monroe (1926)
Professional Bldg., Raleigh, N. C., Op.
Hicks, Wm. J. (1930).....15 King St., Middletown, N. Y., ALR
Higbee, D. R. (1927).....3245 Fourth St., San Diego, Cal., ALR
Higgins, R. P. (1924).....20 Court St., Cortland, N. Y., OALR
*Higgins, S. G.....120 E. Wisconsin Ave., Milwaukee, Wis., OALR
Hilgartner, H. L. (1930).....Norwood Bldg., Austin, Texas, Op.
Hill, C. E. (1919).....160 Bloor St., W., Toronto, Can., Op.
*Hill, Emory (1916).....Professional Bldg., Richmond, Va., Op.
*Hill, Frederick Thayer (1928)
Professional Bldg., Waterville, Me., ALR
*Hill, Howard Foster (1928)
Professional Bldg., Waterville, Me., Op.
Hinman, E. E. (1924)..146 Washington Ave., Albany, N. Y., ALR
Hitz, H. B.....141 E. Wisconsin Ave., Milwaukee, Wis., ALR
*Hobart, Carl (1929).....Metropolitan Bldg., St. Louis, Mo., Op.
Hodge, Geo. E. (1930).....Med. Arts Bldg., Montreal, Can., ALR
Hodson, B. F.....418 Security Bldg., Miami, Fla., OALR
Hoffmann, John N. (1927).....309 Brant Bldg., Canton, O., ALR
*Hoffman, L. G. (1927)....30 N. Michigan Ave., Chicago, Ill., OALR

HOFFMAN, WALTER F. (1920)
817 Summit Ave., Seattle, Wash., Op.
HOFLING, CHAS. A. (1929).....707 Race St., Cincinnati, O., Op.
*HOFMANN, W. P. (1925).....707 Kahl Bldg., Davenport, Ia., OALR
HOGAN, W. L. (1924).....750 Main St., Hartford, Conn., OALR
*HOGSHEAD, J. M.....602 Georgia Ave., Chattanooga, Tenn., OALR
HOGUE, D. W. (1908).....34 W. High St., Springfield, O., OALR
HOHF, JULIUS (1915).....Clinic Bldg., Yankton, S. D., OALR
HOLDSWORTH, FRANK
People's Savings Bank Bldg., Traverse City, Mich., OALR
HOLLOWAY, THOS. B.....1819 Chestnut St., Philadelphia, Pa., Op.
*HOLMES, BENJAMIN H. (1922), 435 Baker Blk., Racine, Wis., ALR
HOLT, E. E., JR.....723 Congress St., Portland, Me., Op.
*HOMPES, J. J.....702 Sharp Bldg., Lincoln, Neb., OALR
*HOOPLE, GORDON D. (1929), Med. Arts Bldg., Syracuse, N. Y., ALR
HOOSS, C. H. A. (1930)....3115 S. Grand Blvd., St. Louis, Mo., Op.
HOPKINS, G. H. (1930).....702 N. Main St., Pueblo, Colo., Op.
HOPKINS, W. E.....321 S. Lorraine Blvd., Los Angeles, Cal., OALR
HORN, W. L. (1929).....1040 Park Ave., New York, N. Y., ALR
*HOURN, GEORGE E. (1920)
511 University Club Bldg., St. Louis, Mo., ALR
HOVDE, A. G. (1916).....Board of Trade Bldg., Superior, Wis., OALR
*HOWARD, C. N. (1919).....Warsaw Clinic, Warsaw, Ind., OALR
HOWARD, E. F. (1919)
First Nat'l Bank Bldg., Vicksburg, Miss., ALR
HOWARD, EDWARD L. (1926)
405 Medical Arts Bldg., Fort Worth, Tex., OALR
*HOWARD, HARVEY J. (1928)
School of Med., Washington University, St. Louis, Mo., Op.
HOWARD, J. W.....Rialto Bldg., Kansas City, Mo., OALR
HOWARD, LLOYD G. (1923)
Council Bluffs Clinic, Council Bluffs, Iowa, OALR
HOWARD, W. H. (1923)
Medical Arts Bldg., Minneapolis, Minn., OALR
*HOWE, A. W. (1922).....Medical Arts Bldg., Tacoma, Wash., OALR
*HOWE, SPENCER S. (1929)
419 Bellingham Nat'l Bank Bldg., Bellingham, Wash., ALR
HUGHES, J. A. (1927).....Sterick Bldg., Memphis, Tenn., OALR
HUGHES, L. W. (1925).....965 Broad St., Newark, N. J., OALR
HUGHES, RAY WHITBECK (1928)
1501 David Whitney Bldg., Detroit, Mich., OALR
HUGHES, T. E. (1922).....1000 W. Grace St., Richmond, Va., ALR
*HUGHES, W. F., 401 Hume-Mansur Bldg., Indianapolis, Ind., Op.
*HUGHES, WENDELL L. (1929)
20 E. 53rd St., New York, N. Y., Op.
HUNT, WESTLEY M. (1929), 33 E. 68th St., New York, N. Y., ALR
*HUNTER, J. F. (1930).....536 Penn Ave., Turtle Creek, Pa., ALR
HUNTER, R. J. (1930).....2011 Chestnut St., Philadelphia, Pa., ALR
HURD, L. M. (1910).....39 E. 50th St., New York City, N. Y., ALR

HURFF, J. W. (1919).....86 Washington St., Newark, N. J., OALR
HURLEY, E. D. (1914).....419 Boylston St., Boston, Mass., Op.
HURST, V. REEVES (1923), 107 E. Methvin St., Longview, Tex., OALR
HUSIK, D. N. (1921).....1930 Chestnut St., Philadelphia, Pa., ALR
HUSSEY, E. J.....245 Maple St., Holyoke, Mass., OALR
HUTCHINSON, ABBOTT TRASK (1928)
30 W. 59th St., New York City, N. Y., ALR
HUTCHISON, K. O.....1509 Sherbrooke St. W., Montreal, Can., ALR

I

*IGLAUER, S.....707 Race St., Cincinnati, O., ALR
*IMPERATORI, C. J.....17 E. 38th St., New York City, N. Y., ALR
INGERMAN, S. M. (1909)
418 Central Park, W., New York City, N. Y., OALR
INGERSOLL, J. M. (1908), 1700 S. Bayshore Lane, Miami, Fla., ALR
INGLIS, HARRY J.....43 Bay State Rd., Boston, Mass., ALR
INGRAM, L. C. (1915).....Orlando Clinic, Orlando, Fla., OALR
IRVIN, EDGAR H.....903 First Nat'l Bk. Bldg., El Paso, Texas, OALR
IRVINE, A. R. (1918), 1144 Roosevelt Bldg., Los Angeles, Cal., Op.
IRVINE, ROBT. S. (1929).....490 Post St., San Francisco, Cal., ALR
IRWIN, V. J., SR.....1634 Main St., Springfield, Mass., OALR
IRWIN, VINCENT J., JR. (1921)
1634 Main St., Springfield, Mass., OALR
ISRAEL, NORMA E. (1919)
2001 Niels Esperon Bldg., Houston, Tex., OALR
ISRAEL, SIDNEY.....2001 Niels Esperon Bldg., Houston, Tex., ALR
IVINS, H. M. (1910)
815 Merchants Nat'l Bank Bldg., Cedar Rapids, Ia., OALR

J

JACKLE, ARTHUR F. (1917), 63 E. Fourth St., Dunkirk, N. Y., Op.
*JACKSON, ATRAS EARL (1928)
Med. Arts Bldg., Fort Worth, Tex., Op.
JACKSON, CHEVALIER.....235 S. 15th St., Philadelphia, Pa., L
JACKSON, J. D. (1920).....232 West 8th St., Erie, Pa., OALR
*JACOBS, MAX W. (1916)
Missouri Theater Bldg., St. Louis, Mo., Op.
JACOBS, S. M.....1018 E. 163rd St., New York City, N. Y., OALR
JACOBY, D. P. A. (1912).....106 Touro St., Newport, R. I., Op.AL
JAQUISH, CHARLES JOSEPH (1928)
904 Westinghouse Bldg., Pittsburgh, Pa., ALR
JARECKY, HERMAN, 168 W. 86th St., New York City, N. Y., OALR
*JARVIS, DEFOREST C. (1929).....107 N. Main St., Barre, Vt., ALR
JEAN, GEO. W.....7 East Valerio, Santa Barbara, Cal., Op.
*JENKINS, M. C.....223 S. Main St., Pratt, Kan., ALR

JOBSON, G. B. (1903)
West Park and Liberty Sts., Franklin, Pa., OALR
*JOHNSON, A. E. (1919) ..... Watertown, S. D., OALR
JOHNSON, H. R. (1913) .... Masonic Temple, Fairmont, W. Va., OALR
JOHNSON, OTIS H. ..... Morehead City, N. C., OALR
JOHNSON, THOMAS HOLLAND (1923)
30 W. 59th St., New York City, N. Y., Op.
JOHNSTON, W. H. (1912)
1421 Chapala St., Santa Barbara, Cal., OALR
JOHNSTON, WILSON (1917) .... 611 Lovejoy St., Portland, Ore., OALR
JONES, C. C. (1924) ..... 19 Garfield Place, Cincinnati, O., ALR
*JONES, CECIL CAIN (1928)
1120 Equitable Bldg., Des Moines, Ia., OALR
JONES, EDLEY HICKS (1928)
First Nat'l Bank Bldg., Vicksburg, Miss., OALR
JONES, EDMUND LLOYD (1926)
Wheeling Clinic, Wheeling, W. Va., OALR
*JONES, E. L. (1913), First Nat'l Bk. Bldg., Cumberland, Md., OALR
JONES, E. W. ..... 44 Atlanta St., Lynn, Mass., Op.A
JONES, F. W. (1919) ..... Times Bldg., Alton, Ill., OALR
*JONES, L. W. (1925) .... 16 N. Goodman St., Rochester, N. Y., OALR
JONES, M. F. (1930) ..... 121 E. 60th St., New York, N. Y., ALR
JONES, U. G. (1927)
Jones Eye, Ear, Nose and Throat Hos., Johnson City, Tenn., ALR
JONES, VINCENT L. (1928)
3511 Washington Ave., St. Louis, Mo., Op.
*JONES, WM. S. (1927), 522 Sheridan Rd., Menominee, Mich., OALR
*JORDAN, GEO. T. (1921), 30 N. Michigan Blvd., Chicago, Ill., OALR
*JOSEPHSON, EMANUEL M. (1929)
993 Park Ave., New York, N. Y., ALR
*JOY, HAROLD HENRY (1926)
504 State Tower Bldg., Syracuse, N. Y., Op.
*JOYNT, A. J. (1924) ..... Black Bldg., Waterloo, Ia., OALR
*JOYNT, M. J. (1930) ..... Royal Bldg., Le Mars, Ia., ALR
JUDD, H. B. (1917) ..... 40 E. 61st St., New York City, N. Y., ALR
*JUDD, J. H. (1930) ..... Medical Arts Bldg., Omaha, Neb., Op.
JUDGE, HARRY V. (1920) ..... 46 Willett St., Albany, N. Y., Op.

K

*KAHN, M. E. (1925) ..... 104 S. Michigan Ave., Chicago, Ill., ALR
KARKEET, ROBERT BUDD (1928)
1203 Selling Bldg., Portland, Ore., OALR
KAUFFMAN, A. B. (1922)
1000 Chimes Bldg., Syracuse, N. Y., ALR
KAUFMAN, A. SPENSER (1917)
1923 Spruce St., Philadelphia, Pa., ALR

KEARBY, D. O. (1917)
709 Hume-Mansur Bldg., Indianapolis, Ind., ALR
KEATING, T. F. (1927)....1042 Maccabee Bldg., Detroit, Mich., OALR
KEEFE, WM. J. (1929), 110 E. Sidney Ave., Mt. Vernon, N. Y., ALR
KEELER, J. C.....Med. Arts Bldg., Philadelphia, Pa., ALR
KEIM, W. F. (1924).....25 Roseville Ave., New York, N. Y., ALR
KEIPER, G. F., JR. (1930)....727 W. 7th St., Los Angeles, Cal., ALR
*KEITH, W. E.....1000 Professional Bldg., Kansas City, Mo., OALR
*KELLER, S. A.....Sioux Falls Clinic, Sioux Falls, S. D., OALR
KELLEY, ISAAC D., JR. (1928)
3720 Washington Blvd., St. Louis, Mo., ALR
KELLY, JOSEPH DOMINIC (1928)
114 E. 54th St., New York City, N. Y., OALR
KEMLER, JOS. (1922).....1908 Eutaw Pl., Baltimore, Md., OALR
KENDALL, M. R. (1919).....Osborn Bldg., Cleveland, O., ALR
*KENNEDY, ELDRED WESTON (1928)
707 Gas and Elect. Bldg., Rochester, N. Y., Op.
KENNON, W. G. (1924).....Doctors' Bldg., Nashville, Tenn., OALR
*KETTELKAMP, FRED O. (1926)
301 Ferguson Bldg., Colorado Springs, Colo., ALR
*KEY, BEN WITT (1914)
100 W. 59th St., New York City, N. Y., Op.
KEY, SAM N.....Norwood Bldg., Austin, Tex., OALR
KEYS, MORTON J. (1921)
100 Jones Blk., Victoria, B. C., Canada, OALR
KIEFER, HUGO A. (1915), Brockman Bldg., Los Angeles, Cal., OALR
*KIEHLE, F. A.....406 Taylor St., Portland, Ore., OALR
KILBOURNE, P. H. (1924)....Fidelity Med. Bldg., Dayton, O., OALR
*KILLEEN, JOHN JOSEPH (1928)
104 S. Michigan Ave., Chicago, Ill., ALR
KINCAID, J. H.....421 W. Church Ave., Knoxville, Tenn., OALR
KINCANNON, LEROY TAYLOR (1928)
The Woodward, Birmingham, Ala., OALR
*KING, CLARENCE (1910), Union Central Bldg., Cincinnati, O., Op.
*KING, CHAS. R. (1920).....Spitzer Bldg., Toledo, O., OALR
*KING, ED.....1002 Carew Tower, Cincinnati, O., ALR
*KING, G. L., SR.....537 E. Market St., Alliance, O., OALR
KING, GEO. L., JR. (1929).....537 Market St., Alliance, O., Op.
KING, J. J. (1916).....30 E. 40th St., New York City, N. Y., ALR
*KING, O. H. (1916)....Medical Arts Bldg., Hot Springs, Ark., OALR
KINNE, SANFORD HALSOY (1928)
73 Main St., Binghamton, N. Y., ALR
*KIRBY, D. B. (1924)....30 W. 59th St., New York City, N. Y., Op.
KIRKPATRICK, SAMUEL.....902 Alabama Ave., Selma, Ala., OALR
KISER, J. D.....Central Nat'l Bank Bldg., Bartlesville, Okla., OALR
KISTLER, O. F.....43 N. Franklin St., Wilkes-Barre, Pa., Op.AR
KISTNER, F. B.....600 Mayer Bldg., Portland, Ore., OALR
KITTELSON, THEODORE N. (1915)
Iverson and Lee Bldg., Fergus Falls, Minn., OALR

KLEINERT, MARGARET NOYES
82 Commonwealth Ave., Boston, Mass., ALR
KLINE, H. G. (1912).....State Tower Bldg., Syracuse, N. Y., ALR
*KLINE, O. R. (1930).....414 Cooper St., Camden, N. J., ALR
KLINEDINST, JOHN FERD. (1906)
220 S. George St., York, Pa., Op.A
KLINGNER, THOMAS O. (1920)
318 Landers Bldg., Springfield, Mo., OALR
*KNAPP, ARNOLD.....10 E. 54th St., New York City, N. Y., Op.
KNAPP, B. J.....Citizen's Bank Bldg., Evansville, Ind., OALR
KNAPP, F. N. (1925).....Bradley Bldg., Duluth, Minn., OALR
KNAUER, WM. J. (1930).....Buckman Bldg., Jacksonville, Fla., ALR
*KNEEDLER, G. C. (1912)....Professional Bldg., Pittsburgh, Pa., ALR
*KNODE, A. R. (1916).....708 Med. Arts Bldg., Omaha, Neb., ALR
KNOWLTON, CHAS. D. (1929)
520 Commonwealth Ave., Boston, Mass., ALR
KOEBBE, EZRA EDWIN (1929).....Columbus, Neb., ALR
*KOENIG, IVAN J. (1928).....40 North St., Buffalo, N. Y., Op.
*KOOBS, H. J. G. (1920)....117½ W. Walnut St., Rogers, Ark., OALR
*KOPETZKY, S. J.....51 W. 73rd St., New York City, N. Y., ALR
*KRAFT, OSCAR H.....1518 N. Dearborn Pkwy., Chicago, Ill., Op.
KRAUSS, FREDERICK.....1930 Chestnut St., Philadelphia, Pa., OALR
KREBS, ADOLPH (1914).....Jenkins Arcade, Pittsburgh, Pa., Op.
KRESS, GEO. H.....245 Bradbury Bldg., Los Angeles, Cal., OALR
KREUTZ, G. C. (1930)....Henry Ford Hospital, Detroit, Mich., ALR
*KRIEGER, G. M.....701 Washington St., Michigan City, Ind., OALR
*KRIEGER, WM. A. (1919), 35 Market St., Poughkeepsie, N. Y., OALR
KRIMSKY, JOSEPH (1928)
263 Eastern Parkway, Brooklyn, N. Y., OALR
KRUG, ERNEST F. (1908)
12 W. 44th St., New York City, N. Y., Op.
KRUSKAL, I. D. (1925).....863 Prospect Pl., Brooklyn, N. Y., Op.
*KUHN, H. S. (1930).....First Trust Bldg., Hammond, Ind., Op.
*KUHN, HUGH A. (1928)....First Trust Bldg., Hammond, Ind., ALR
KULLY, B. M. (1922).....Med. Arts Bldg., Omaha, Neb., ALR

L

LAESSLE, HENRY A. (1922)
The Lenox, 13th and Spruce Sts., Philadelphia, Pa., ALR
*LAFF, H. I. (1930).....Metropolitan Bldg., Denver, Colo., ALR
LA FORCE, B. D.....1140 Beverley Way, Altadena, Cal., OALR
*LA FORCE, E. F. (1908)
Iowa State Bank Bldg., Burlington, Ia., OALR
LAMB, HARVEY D. (1920)
826 Metropolitan Bldg., St. Louis, Mo., Op.
*LAMB, HARRY H. (1929)
American Bank Bldg., Davenport, Ia., ALR

LAMB, ROBT. SCOTT.....Stoneleigh Ct., Washington, D. C., Op.
*LANCASTER, W. B. (1913)
520 Commonwealth Ave., Boston, Mass., Op.
LANDES, GEORGE A.....Pampa, Tex., OALR
LANDFRIED, C. J.....607 Baronne Bldg., New Orleans, La., ALR
LANDIS, J. E.....517 Walnut St., Reading, Pa., ALR
LANING, J. H. (1918).....907 Rialto Bldg., Kansas City, Mo., ALR
LAPSLEY, ROBERT M. (1902)....Masonic Temple, Keokuk, Ia., OALR
*LARGE, S. H. (1904).....Hanna Bldg., Cleveland, O., ALR
*LARKIN, BERNARD J. (1916)
Hume-Mansur Bldg., Indianapolis, Ind., Op.
LARSEN, CARL L. (1916)
1027 Lowry Medical Bldg., St. Paul, Minn., OALR
*LARUE, C. L. (1922).....Highland Clinic, Shreveport, La., OALR
LASSALLE, ALBERT (1924), 3439 St. Denis St., Montreal, Can., OALR
LASSEN, FRITZ (1915).....Pueblo Clinic, Pueblo, Colo., OALR
LA VIGNE, A. A.....471 Park Ave., New York City, N. Y., OALR
LAWRENCE, GRANVILLE A.
Medical Arts Bldg., Philadelphia, Pa., Op.
*LAWWILL, STEWART (1928)
Medical Arts Bldg., Chattanooga, Tenn., OALR
LEA, JOS. A.....3526 Travis St., Fort Worth, Tex., ALR
LEAVENWORTH, R. O. (1930)
Lowry Medical Arts Bldg., St. Paul, Minn., Op.
LEAVY, C. A.....Metropolitan Bldg., St. Louis, Mo., ALR
*LEBENSOHN, JAMES E. (1927)
25 E. Washington St., Chicago, Ill., OALR
LE COMPTE, E. D. (1927)
627 Boston Bldg., Salt Lake City, Utah., OALR
*LEDERER, FRANCIS L. (1925)
30 N. Michigan Blvd., Chicago, Ill., ALR
LEECH, J. W.....369 Broad St., Providence, R. I., OALR
LEECH, V. M. (1930).....55 E. Washington St., Chicago, Ill., Op.
LEFLER, ANNA B.(1918), 610 S. Broadway, Los Angeles, Cal., OALR
*LEHAN, JAMES W.....Park Place Bldg., 8th St., Greeley, Colo., OALR
*LEJEUNE, F. E. (1927)
632 Maison Blanche Bldg., New Orleans, La., ALR
LEMERE, H. B. (1912)
607 Hollywood Security Bldg., Hollywood, Cal., OALR
LEMOINE, A. N. (1920).....Rialto Bldg., Kansas City, Mo., Op.
LENKER, JOHN N. (1908).....1020 Huron Road, Cleveland, O., ALR
*LENT, E. J.....122 N. Lafayette Blvd., South Bend, Ind., OALR
LERNER, MACY LEVI(1923), 332 Park Ave., Rochester, N. Y., Op.
*LESTER, HARRY S. (1909).....420 Murray Bldg., Streator, Ill., OALR
*LEVENGOOD, H. W. (1927)
327 Wilshire Blvd., Santa Monica, Cal., OALR
LEVY, LOUIS (1916).....1052 Madison Ave., Memphis, Tenn., ALR
LEVY, ROBERT (1896).....Metropolitan Bldg., Denver, Colo., ALR
*LE WIN, THURBEP (1929), 112 Linwood Ave., Buffalo, N. Y., Op.

*Lewis, A. C. (1916).....Exchange Bldg., Memphis, Tenn., OALR
Lewis, Eugene R. (1926), Roosevelt Bldg., Los Angeles, Cal., ALR
Lewis, Fielding O.....259 S. 17th St., Philadelphia, Pa., ALR
*Lewis, F. Park.....454 Franklin St., Buffalo, N. Y., Op.
Lewis, George A. (1920).....Wall Bldg., Roundup, Mont., OALR
Lewis, J. D. (1921).....1534 State St., Santa Barbara, Cal., OALR
Lewis, J. P. (1915).....543 Boylston St., Boston, Mass., ALR
Lewis, Philip Meriwether (1928)
Exchange Bldg., Memphis, Tenn., OALR
Lewis, William W. (1921)
836 Lowry Bldg., St Paul, Minn., OALR
*Leyda, James H. (1927), Metropolitan Bldg., Denver, Colo., ALR
*Lichtenberg, J. S. (1906).....Rialto Bldg., Kansas City, Mo., Op.
Lidikay, C. J. (1920).....903 N. 7th St., Kansas City, Kan., OALR
*Lierle, Dean McAllister (1928)
University Hospital, Iowa City, Ia., ALR
*Lifschutz, Jacob (1925), 310 S. Michigan Ave., Chicago, Ill., OALR
Lillie, Harold I.....Mayo Clinic, Rochester, Minn., ALR
Lillie, W. Ivan (1930).....Mayo Clinic, Rochester, Minn., Op.
*Lingeman, B. N. (1930)
Ben Hur Bldg., Crawfordsville, Ind., ALR
*Lingeman, Ed. L. (1929)
406 Pennway Bldg., Indianapolis, Ind., ALR'
*Linn, J. G. (1919), 7026 Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
Lischkoff, M. A. (1913).....Blount Bldg., Pensacola, Fla., OALR
Litchfield, J. T. (1921)
Medical Arts Bldg., Minneapolis, Minn., OALR
Livingston, Geo. S. (1930), 185 N. Wabash Ave., Chicago, Ill., ALR
Lloyd, A. W. (1915).....804 Lloyd Bldg., Hammond, Ind., OALR
*Lloyd, Ralph I. (1925).....14 Eighth Ave., Brooklyn, N. Y., Op.
Loeb, Clarence (1907)
1054 Tower Rd., Hubbard Woods, Ill., Op.
Loftus, John Edward (1921)
1930 Chestnut St., Philadelphia, Pa., ALR
Lokey, H. M.....Medical Arts Bldg., Atlanta, Ga., OALR
Lombardo, Melchiore (1923)
142 Joralemon St., Brooklyn, N. Y., Op.
Loomis, E. A. (1916)
Medical Arts Bldg., Minneapolis, Minn., OALR
Looper, Edward Anderson (1924)
104 Madison St., Baltimore, Md., OALR
Lorie, Alvin J. (1920).....Commerce Bldg., Kansas City, Mo., ALR
Loring, J. B.....25 E. Washington St., Chicago, Ill., OALR
Love, John G. (1920).....Ilgenfritz Bldg., Sedalia, Mo., OALR
*Lowry, B. W. (1925).....205 Zack St., Tampa, Fla., OALR
Lubman, Max (1919), 17 E. 96th St., New York City, N. Y., ALR
Lucas, Harold Roy (1928)
Medical Dental Bldg., Portland, Ore., OALR
*Luedde, W. H. (1916).....Metropolitan Bldg., St. Louis, Mo., Op.

LUKENS, CHARLES (1906).....316 Michigan St., Toledo, O., OALR
*LUKENS, J. A. (1930).....316 Michigan St., Toledo, O., ALR
LUKENS, ROBT. McD.....1923 Spruce St., Philadelphia, Pa., ALR
LUNDON, A. E. (1927)....1501 Crescent St., Montreal, Canada, ALR
LUPTON, IRVING MARTIN (1928)
Medical Arts Bldg., Portland, Ore., OALR
LURIE, M. H. (1930).....483 Beacon St., Boston, Mass., ALR
LUX, PAUL (1925).....409 Commerce Bldg., Kansas City, Mo., ALR
*LYLE, DONALD J. (1927).....19 W. 7th St., Cincinnati, O., OALR
*LYMAN, HARRY W.(1923), 906 Carleton Bldg., St. Louis, Mo., ALR
*LYNCH, R. C. (1913)
Maison Blanche Bldg., New Orleans, La., ALR
*LYONS, H. R.....30 N. Michigan Blvd., Chicago, Ill., ALR

M

MCALESTER, ANDREW W., JR.
Rialto Bldg., Kansas City, Mo., Op.
MCALLISTER, J. C. (1910).....10 S. Mill St., Ridgway, Pa., AL
*MCBEAN, GEO. M.....58 E. Washington St., Chicago, Ill., OALR
*MCBRIDE, W. O. (1917)
2902 S. Fairfield Ave., Ft. Wayne, Ind., OALR
*MCCANNEL, A. D.....33 Central Ave. W., Minot, N. D., OALR
MCCARTNEY, OLIVER P. (1920)
Valley Bank Bldg., Mesa, Ariz., OALR
MCCARTY, VIRGIL W.....Professional Bldg., Kansas City, Mo., OALR
*MCCASKEY, C. H.....Continental Bk. Bldg., Indianapolis, Ind., ALR
*MCCAW, JOHN A.....418 Majestic Bldg., Denver, Colo., Op.
MCCLELLAN, W. E. (1927)
50 Farmington Ave., Hartford, Conn., OALR
MCCLELLAND, CARL C. (1923)
1857 David Whitney Bldg., Detroit, Mich., OALR
MCCONNELL, T. E., Logan Trust Bldg., New Kensington, Pa., OALR
MCCOOL, J. L. (1911).....450 Sutter St., San Francisco, Cal., Op.
*MCCORMICK, T. F. (1930)
120 E. Wisconsin Ave., Milwaukee, Wis., ALR
MCCOY, J.....730 Fifth Ave., New York City, N. Y., ALR
MCCOY, L. L. .....817 Summit Ave., Seattle, Wash., Op.
MCCREADY, J. H.....121 University Pl., Pittsburgh, Pa., ALR
*MCCULLOCH, R. J. P. (1920)
830 Medical Arts Bldg., Toronto, Can., OALR
*MCCULLOUGH, C. J. (1929)
Washington Trust Bldg., Washington, Pa., ALR
*MCDANNALD, C. E. (1927)
100 W. 59th St., New York City, N. Y., Op.
MCDAVITT, BERTHA SCHENCK STECKER (1929)
Woodson Eye, Ear, Nose and Throat Hosp., Temple, Texas, ALR
*MCDERMOTT, JOHN J. (1924)
1821 Niles Ave., St. Joseph, Mich., OALR

McDIARMID, H. OLIVER (1918)
35 Clement Block, Brandon, Manitoba, Can., OALR
McDONALD, C. L.....964 Rose Bldg., Cleveland, O., OALR
McDONALD, DENNIS J. (1926)
17 E. 38th St., New York City, N. Y., ALR
*McDOWELL, N. D. (1914)
275 Alexander St., Rochester, N. Y., OALR
McFARLANE, MURRAY.....190 Bloor St. E., Toronto, Can., OALR
*McGARVEY, WM. ED. (1929)
Jackson City Bank, Jackson, Mich., ALR
McGILLICUDDY, O. B. (1930)
Metropolitan Bldg., Denver, Colo., ALR
*McGINNIS, EDWIN.....104 S. Michigan Blvd., Chicago, Ill., ALR
McGIVERN, C. S. (1922)
101 S. Indiana Ave., Atlantic City, N. J., ALR
*McGUIRE, H. H.....105 N. Braddock St., Winchester, Va., OALR
*McHENRY, D. D. (1912)
604 Medical Arts Bldg., Oklahoma City, Okla., OALR
McHENRY, L. C. (1930)
117 N. Broadway, Oklahoma City, Okla., ALR
McKEE, CLAUD W. (1917)....Coulter Bldg., Greensburg, Pa., OALR
McKEE, GEO. J. (1922), Westinghouse Bldg., Pittsburgh, Pa., ALR
McKEE, JOSEPH WALLACE (1928)
1222 Rialto Bldg., Kansas City, Mo., Op.
McKEE, OTTO S.....83 Bryant St., Buffalo, N. Y., ALR
*McKEE, S. HANFORD (1910)
1528 Crescent St., Montreal, Can., Op.
*McKELVEY, A. D.....160 Bloor St. W., Toronto, Can., ALR
McKENNAN, JAS. W.
514 Washington Trust Bldg., Washington, Pa., Op.
McKEOWN, ELMER F.....Republic Bldg., Denver, Colo., OALR
*McKINNEY, A. R. (1922)
704 Second Nat'l Bk. Bldg., Saginaw, Mich., OALR
McKINNEY, R.....899 Madison Ave., Memphis, Tenn., ALR
McLAUGHLIN, P. A.
71 S. Washington St., Wilkes-Barre, Pa., OALR
McLAURIN, JOHN G. (1926)
Medical Arts Bldg., Dallas, Tex., OALR
McLOONE, JOHN J. (1926)..611 Heard Bldg., Phoenix, Ariz., OALR
*McMAHON, BERNARD JOHN (1926)
Missouri Theater Bldg., St. Louis, Mo., ALR
*McMURRAY, J. B., Washington Trust Bldg., Washington, Pa., OALR
*McNALLY, WILLIAM JAMES
Medical Arts Bldg., Montreal, Canada, ALR
*McNICHOLS, W. A. (1925)
Dixon Nat'l Bank Bldg., Dixon, Ill., OALR
*McRAE, JOHN H.....Grand Rapids Clinic, Grand Rapids, Mich., Op.
McREYNOLDS, G. S.....Professional Bldg., Temple, Texas, ALR

McREYNOLDS, JOHN O.
907 Mercantile Bank Bldg., Dallas, Tex., OALR
*McWILLIAMS, CHAS. A. (1927)
G. and S. I. Bldg., Gulfport, Miss., ALR
MACDONALD, J. C. (1930)
301 W. 12th St., Oklahoma City, Okla., ALR
MACFARLANE, P. B., 152 James St., S. Hamilton, Ont., Can., OALR
MACKENZIE, ALICE V. (1919)
1724 Spruce St., Philadelphia, Pa., OALR
MACKENZIE, GEO. W. (1912)
1724 Spruce St., Philadelphia, Pa., OALR
*MACLACHLAN, A. A.....May Bldg., Pittsburgh, Pa., ALR
MACMILLAN, J. A. (1925), 1410 Stanley St., Montreal, Can., Op.
MACNIE, J. S.....74 S. 11th St., Minneapolis, Minn., OALR
MADDEN, REED.....Allen Bldg., Xenia, O., OALR
MAGGARD, D. I. (1920).....715 Orpheum Bldg., Wichita, Kan., OALR
MAGHY, CHAS. A. (1926)
Wilshire Medical Bldg., Los Angeles, Cal., Op.
MAHONEY, PAUL LEO (1928)
Donaghey Bldg., Little Rock, Ark., ALR
MAIDEN, S. D. (1918), 403 Bennett Bldg., Council Bluffs, Ia., OALR
MAIRE, LEWIS E. (1906)....710 Kresge Bldg., Detroit, Mich., OALR
*MALONE, F. B. (1930), Lubbock Sanitarium, Lubbock, Texas, ALR
MALONEY, THOS. J.....1032 Lowry Bldg., St. Paul, Minn., OALR
*MANN, B. H. (1930)
N. E. corner 20th and Chestnut Sts., Philadelphia, Pa., Op.
MANN, R. H. T. (1913)
State Nat'l Bank Bldg., Texarkana, Texas, OALR
*MARCH, CLARA A. (1922), 465 Ashland Ave., Buffalo, N. Y., Op.
MARKEL, J. CLYDE.....Westinghouse Bldg., Pittsburgh, Pa., Op.
*MARKS, JULIAN B. (1929)
1st Nat'l Bank Bldg., Milwaukee, Wis., ALR
*MARKS, SAMUEL BLACKBURN (1928)
Sec. Trust Bldg., Lexington, Ky., ALR
*MARLOW, SEARLE B. (1924)
1004 State Tower Bldg., Syracuse, N. Y., Op.
*MARQUIS, GEO. P.....30 N. Michigan Blvd., Chicago, Ill., ALR
MARSDEN, W. C. (1930), 64 N. Franklin St., Wilkes-Barre, Pa., ALR
MARSHALL, GEORGE GUERIN.....Gryphon Bldg., Rutland, Vt., OALR
MARSHALL, WATSON.....121 University Pl., Pittsburgh, Pa., ALR
*MARTIN, ROY D. (1929).....Foote Bldg., Hastings, Neb., ALR
MARTIN, W. O., JR.....Medical Arts Bldg., Atlanta, Ga., Op.
*MASON, ROY EARL (1928).....Frisco Bldg., St. Louis, Mo., Op.
*MASTERS, ROBERT JOHN (1928)
Hume-Mansur Bldg., Indianapolis, Ind., Op.
*MATHENY, R. C.....306 E. Main St., Galesburg, Ill., OALR
MATHESON, J. P. (1915).....6 West 7th St., Charlotte, N. C., ALR
MATTHEWS, JUSTUS..Medical Arts Bldg., Minneapolis, Minn., LR

MAULDIN, L. O.
Chamber of Commerce Bldg., Greenville, S. C., OALR
MAUMENEE, A. E.....711 Empire Bldg., Birmingham, Ala., OALR
MAXEY, E. E. (1911).....609 Becker Bldg., Aberdeen, Wash., OALR
*MAY, CHAS. H. (1904)
698 Madison Ave., New York City, N. Y., Op.
*MAY, J. V. (1915).....1703 Main St., Marinette, Wis., OALR
MAYBAUM, J. L. (1916)
1079 Park Ave., New York City, N. Y., ALR
*MAYER, LEO LOEB (1928)
104 S. Michigan Ave., Chicago, Ill., Op.
MAYERBERG, EMIL R. (1929)
Medical Arts Bldg., Wilmington, Del., ALR
MEANOR, WM. C.....619 Westinghouse Bldg., Pittsburgh, Pa., Op.
*MEANS, R. G. (1930).....137 East State St., Columbus, O., ALR
MEIERHOF, E. L. (1912), 12 E. 86th St., New York City, N.Y., OALR
MELLINGER, WM. J. (1927)
120 E. Sola St., Santa Barbara, Cal., ALR
MENGER, LOUIS C. (1929), 202 Weirfield St., Brooklyn, N. Y., ALR
*MERCER, RAY (1920).....W. C. U. Bldg., Quincy, Ill., OALR
*MERCER, R. E.....David Whitney Bldg., Detroit, Mich., LR
MERICA, F. W. (1930).....14805 Detroit Ave., Lakewood, O., ALR
MERKEL, CHARLES CARSON (1928)
David Whitney Bldg., Detroit, Mich., ALR
MERRILL, H. G. (1917)
35 East 2nd South St., South, Provo, Utah, OALR
MERRILL, W. HOWE (1906)
Bay State Bldg., Lawrence, Mass., OALR
*MERTINS, PAUL STEARNS (1928)
1115 Bell Bldg., Montgomery, Ala., OALR
*MESSIER, A. E. (1925).....390 Main St., Worcester, Mass., OALR
MESSINGER, H. C.....210 Angell St., Providence, R. I., Op.
METZ, ROY B.....1214 Guardian Bldg., Cleveland, O., Op.
METZENBAUM, MYRON (1912).....Rose Bldg., Cleveland, O., ALR
*MEYER, SAMUEL JAMES (1928)
58 E. Washington St., Chicago, Ill., Op.
*MIDDLETON, A. B.....Sterry Block, Pontiac, Ill., OALR
*MIDDLETON, HARRY E. (1920)....103 E. Broadway, Alton, Ill., OALR
MILLER, ALICE R. (1919)
418 Fern St., West Palm Beach, Fla., OALR
MILLER, C. E.....917 E. Main St., Munsie, Ind., OALR
MILLER, CLIFTON M...Stuart Circle Hospital, Richmond, Va., OALR
MILLER, E. B.....2008 Walnut St., Philadelphia, Pa., Op.
MILLER, H. ED. (1930).....Missouri Bldg., St. Louis, Mo., ALR
MILLER, HERMAN B. (1928)
810 Metropolitan Bldg., St. Louis, Mo., ALR
*MILLER, HUGH.....Chambers Bldg., Kansas City, Mo., OALR
MILLER, JOHN.....121 East 60th St., New York, N. Y., ALR
MILLER, J. W. (1929).....305 E. 17th St., New York, N. Y., ALR

*MILLETTE, J. W.....Reibold Bldg., Dayton, O., OALR
MINER, S. G. (1896).....2641 E. Jefferson, Detroit, Mich., ALR
MINOR, C. L.....First Nat'l Bank Bldg., Springfield, O., OALR
MINOR, H. F.....Randolph Bldg., Memphis, Tenn., OALR
MINSKY, HENRY (1926)
1 West 86th St., New York City, N. Y., Op.
MINTON, WM. H.....First State Bank Bldg., St. Joseph, Mo., OALR
MITCHELL, E. K. (1915)
710 W. Lehigh Ave., Philadelphia, Pa., ALR
*MITCHELL, H. E.....15701 Detroit Ave., Lakewood O., ALR
*MITHOEFER, W.....Doctors Bldg., Cincinnati, O., ALR
MITTENDORF, A. D.....339 Park Ave., New York City, N. Y., Op.
MITTENDORF, W. KEITH
115 E. 53rd St., New York City, N. Y., Op.
*MOISE, ALLYN B. (1918), Medical Arts Bldg., Shreveport, La., ALR
*MOLT, WM. F. (1911)
529 Bankers Trust Bldg., Indianapolis, Ind., ALR
MONAGHAN, D. G.....Majestic Bldg., Denver, Colo., Op.
MONAHAN, J. A. (1913).....181 Chestnut St., Clinton, Mass., OALR
MONOSMITH, O. B. (1912).....O. P. S. Bldg., Lorain, O., OALR
*MONSON, S. H.....1322 B. F. Keith Bldg., Cleveland, O., OALR
*MONTGOMERY, DANIEL CAMERON (1928)
Greenley Bldg., Greenville, Miss., ALR
MOORE, G. A. (1915).....353 Main St., Palmer, Mass., OALR
MOORE, GEORGE H. (1919)
18th and Chestnut Sts., Philadelphia, Pa., OALR
MOORE, JOHN D. (1925)....6 Washington St., Bloomfield, N. J., OALR
*MOORE, P. G. (1930).....Medical Arts Bldg., Cleveland, O., Op.
MOORE, PAUL MCNARY, JR.
University Hospital, Iowa City, Ia., ALR
MOORE, ROY SEELEY (1928)
Medical Arts Bldg., Syracuse, N. Y., ALR
*MOORE, R. B. (1930).....Trinity Hospital, Little Rock, Ark., ALR
*MOORE, T. K.....Second Nat'l Bank Bldg., Akron, O., OALR
MOORE, T. W. (1901)....1050 Fifth Ave., Huntington, W. Va., OALR
MOORE, WILLIAM FREDERICK
255 S. 16th St., Philadelphia, Pa., ALR
MORGAN, JAMES A. (1916)
48 Young Bldg., Honolulu, T. Hawaii, OALR
MORIARTY, RICHARD W. (1928)
Medical Center, White Plains, N. Y., OALR
*MORLEY, G. A. (1930)
Crookston State Bank Bldg., Crookston, Minn., Op.
MORRIS, A. G. (1918).....277 Alexander St., Rochester, N. Y., ALR
MORRISON, WM. WALLACE (1926)
39 E. 50th St., New York City, N. Y., ALR
*MORSE, A. W.....507 Phoenix Bldg., Butte, Mont., OALR
*MORSMAN, L. W. (1920)....Morsman Clinic, Hibbing, Minn., Op.

MORTON, HOWARD McI.
Insurance Exchange Bldg., Minneapolis, Minn., Op.
MORTON, J. B. (1924).....90 E. Warren Ave., Detroit, Mich., OALR
*MOSHER, HARRIS P.....828 Beacon St., Boston, Mass., ALR
MOSS, HARVEY E.....Luhrs Bldg., Phoenix, Ariz., OALR
MOTT, JOHN S.....214 Argyle Bldg., Kansas City, Mo., OALR
*MOTTO, M. P. (1929).....Rose Bldg., Cleveland, O., Op.
MOULTON, EVERETT C.
Merchants Nat'l Bank Bldg., Ft. Smith, Ark., OALR
MOULTON, H. (1900)
Merchants Nat'l Bank Bldg., Ft. Smith, Ark., OALR
*MULLIN, WM. V. (1913)
Cleveland Clinic, Euclid Ave. at 93rd St., Cleveland, O., ALR
*MULSOW, J. E. (1921)
221 Wisconsin Ave. W., Milwaukee, Wis., OALR
*MUNCASTER, S. B.....1026 16th St. N. W., Washington, D. C., Op.
MUNCY, WM. M. (1914).....162 Angell St., Providence, R. I., OALR
*MUNDT, G. H. (1912).....25 E. Washington St., Chicago, Ill., OALR
*MUNSON, F. I. (1930).....2201 E. Jefferson Ave., Detroit, Mich., ALR
*MURPHY, A. B. (1930).....2830 Grand Ave., Everett, Wash., ALR
MURPHY, J. G.....Murchison Bldg., Wilmington, N. C., OALR
*MURRAY, A. N.....4753 Broadway, Chicago, Ill., OALR
MUSSUN, W. G. (1924).....Hanna Bldg., Cleveland, O., ALR
MYERS, ELMER A., 407 Board of Trade Bldg., Superior, Wis., OALR
MYERS, EUGENE LEE (1926), 207 Wall Bldg., St. Louis, Mo., ALR
*MYERS, JOHN L. (1920), 815 Shukert Bldg., Kansas City, Mo., OALR
MYERSON, M. C. (1923).....12 E. 86th St., New York, N. Y., ALR

N

*NADEAU, E. G.....Bellin Bldg., Green Bay, Wis., OALR
NAFTZGER, J. B.....Davidson Bldg., Sioux City, Ia., OALR
*NANCE, WILLIS O.....30 N. Michigan Blvd., Chicago, Ill., OALR
NASH, C. STEWART (1929)
26 S. Goodman St., Rochester, N. Y., ALR
NEEPER, EDW. R.
Ex. Nat'l Bank Bldg., Colorado Springs, Colo., Op.
*NEHER, EDWIN MANSON (1926)
Boston Bldg., Salt Lake City, Utah, Op.
NEIGHBORS, CLARENCE A.
120 S. Los Angeles St., Anaheim, Cal., OALR
NELSON, CHAS. F.....606 Schofield Bldg., Cleveland, O., OALR
NELSON, LEO A. (1930).....4105 Live Oak St., Dallas, Texas, ALR
*NELSON, LOUIS A.....830 Lowry Bldg., St. Paul, Minn., Op.
*NELSON, LLOYD L. (1923).....Fleming Bldg., Norfolk, Neb., OALR
NEMOURS, PAUL R. (1929).....3852 Greer Ave., St. Louis, Mo., ALR
NEUMANN, WILLIAM HENRY (1928)
1005 N. 8th St., Sheboygan, Wis., OALR

NEW, GORDON B.....Mayo Clinic, Rochester, Minn., L
NEWCOMB, JOHN R.
410 Hume-Mansur Bldg., Indianapolis, Ind., Op.
NEWELL, WM. S.....1029 Vermont Ave., Washington, D. C., Op.
*NEWHART, HORACE (1905)
Medical Arts Bldg., Minneapolis, Minn., ALR
*NEWTON, F. H. (1925)....907 Mercantile Bldg., Dallas, Texas, OALR
NEWTON, L. A. (1919)
609 Medical Arts Bldg., Oklahoma City, Okla., OALR
NISSELSON, MAX.....754 Beck St., New York City, N. Y., ALR
NOONAN, W. J. (1905).....4753 Broadway, Chicago, Ill., OALR
NORCROSS, E. P. (1924).....8 S. Michigan Ave., Chicago, Ill., ALR
NORTHCOTT, THOMAS A. (1926)
17 E. 38th St., New York City, N. Y., Op.
*NOVAK, FRANK J., JR.....30 N. Michigan Blvd., Chicago, Ill., ALR
NUGENT, OSCAR B. (1930)
231 W. Washington St., Chicago, Ill., Op.
NUTTER, C. F.....16 Amherst St., Nashua, N. H., Op.

O

OAST, SAMUEL P., JR. (1929)
30 E. 40th St., New York, N. Y., Op.
*OBERG, C. M.....Syndicate Bldg., Minneapolis, Minn., OALR
*O'BRIEN, C. S. (1924).....University Hospital, Iowa City, Ia., Op.
O'BRIEN, G. R. (1930)....307 Sterling Place, Brooklyn, N. Y., ALR
*O'BRIEN, STEVE A. (1916)
1st Nat'l Bank Bldg., Mason City, Ia., OALR
O'CONNOR, D. F. (1918).....671 Broad St., Newark, N. J., OALR
*O'CONNOR, RODERICK P.....1904 Franklin St., Oakland, Cal., Op.
ODENEAL, THOMAS H. (1929)....234 Cabot St., Beverly, Mass., OALR
OERTEL, T. E.....S. F. Bldg., Augusta, Ga., OALR
OESTERLING, H. E. (1922)
642 N. Interlachen Ave., Winter Park, Fla., OALR
OFFUTT, W. N. (1905)
406 Security Trust Bldg., Lexington, Ky., OALR
OLMSTEAD, WM. DEF. (1930)
1920 Pacific Ave., Atlantic City, N. J., Op.
OLSHO, S. L. (1912).....235 S. 15th St., Philadelphia, Pa., OALR
*OPPEGAARD, C. L. (1930)..220 S. Broadway, Crookston, Minn., ALR
*ORCUTT, D. C. (1912).....30 N. Michigan Ave., Chicago, Ill., Op.
O'REILLY, CHARLES A. (1911)
521 Medical Arts Bldg., Philadelphia, Pa., ALR
*O'REILLY, W. F. (1913).....44 S. Common St., Lynn, Mass., OALR
*O'ROURKE, D. H. (1927)....217 Imperial Bldg., Denver, Colo., Op.
ORR, ADRIAN V. B. (1929).....525 Welsh St., Chester, Pa., ALR
*ORTON, HENRY B.....24 Commerce St., Newark, N. J., ALR
O'SULLIVAN, T. J. (1927).....10 Pine St., Portland, Me., ALR

OVERMAN, F. V.....Hume-Mansur Bldg., Indianapolis, Ind., ALR
OWEN, F. S.....Medical Arts Bldg., Omaha, Neb., OALR

P

*PACE, ERRETT (1930)....City Nat'l Bank Bldg., Paducah, Ky., ALR
*PACKARD, LOUIS A. (1926)
Haberfelde Bldg., Bakersfield, Cal., OALR
*PAINTER, A. M. (1918)..Professional Bldg., Kansas City, Mo., ALR
PALMER, ARTHUR (1926)
667 Madison Ave., New York City, N. Y., ALR
PALMER, BASCOM HEADEN (1928)
502 Huntington Bldg., Miami Beach, Fla., OALR
*PALMER, EDW. M. (1919)....629 Beacon Bldg., Wichita, Kan., OALR
PALMER, F. E. (1928).....301 Main St., Sterling, Colo., OALR
PARK, J. WALTER (1910), 32 N. Second St., Harrisburg, Pa., OALR
*PARKER, WALTER R.....David Whitney Bldg., Detroit, Mich., Op.
PARRAMORE, JAMES B. (1929)
St. James Bldg., Jacksonville, Fla., ALR
PARRISH, ROBERT E.....Medical Arts Bldg., San Antonio, Tex., ALR
PARSONS, J. G.....Crookston Clinic, Crookston, Minn., OALR
PATTERSON, ELLEN J. (1917)
121 University Pl., Pittsburgh, Pa., ALR
*PATTERSON, W. E.....74 S. 11th St., Minneapolis, Minn., OALR
PAUL, WM. C. (1930).....Washington Bldg., Tacoma, Wash., ALR
*PAYNE, A. L. (1908).....133 S. Barston St., Eau Claire, Wis., OALR
PAYNE, R. J. (1925)....University Club Bldg., St. Louis, Mo., ALR
PAYNE, V. LEROY.....1018 Madison Ave., Memphis, Tenn., ALR
*PEARLMAN, S. J.....180 N. Michigan Ave., Chicago, Ill., ALR
*PEARSON, WM. W. (1908)
417 Bankers Trust Bldg., Des Moines, Ia., OALR
PEELER, C. N. (1920).....6 W. 7th St., Charlotte, N. C., OALR
PEERY, VANCE PRICE (1929).....Kinston, N. C., ALR
PEIRCE, HOWARD W. (1923)
704 Medical Arts Bldg., Detroit, Mich., ALR
*PELZ, MORT D. (1929).....Missouri Bldg., St. Louis, Mo., ALR
*PEMBER, AUBREY H. (1926)
500 W. Milwaukee St., Janesville, Wis., OALR
PENDEXTER, RALPH S. (1926)
1424 K St., N. W., Washington, D. C., Op.
PENTECOST, R. S. (1926).....184 Bloor St., Toronto, Canada, OALR
PERSKY, A. H. (1930).....1922 Spruce St., Philadelphia, Pa., ALR
*PETER, L. C. (1914).....1930 Chestnut St., Philadelphia, Pa., Op.
PETERMAN, H. E. (1912)....518 Cathedral St., Baltimore, Md., OALR
PETERSON, R. A. (1920)
150 Fifth Ave., New York City, N. Y., OALR
PFAFFLIN, C. A., 445 Bankers Trust Bldg., Indianapolis, Ind., OALR
PFINGST, A. O.....Heyburn Bldg., Louisville, Ky., Op.

PFINGSTEN, C. F. (1916)
6370 Waterman Ave., St. Louis, Mo., ALR
PHELPS, KENNETH A. (1920)
730 La Salle Bldg., Minneapolis, Minn., OALR
PHILLIPS, WM. LINTON....131 Linwood Ave., Buffalo, N. Y., Op.
PHINNEY, F. D.....22 W. 7th St., Cincinnati, O., OALR
PINKERTON, F. J. (1929)
46 Young Hotel Bldg., Honolulu, Hawaii, OALR
*PIPER, ARTHUR SYLVESTER (1928), 610 S. Monroe, Enid, Okla., OALR
PITKIN, C. E.....Osborn Bldg., Cleveland, O., ALR
*PLUMER, JOHN SHAFFER (1928)
121 University Pl., Pittsburgh, Pa., Op.
*POLLOCK, H. L. (1911).....2551 N. Clark St., Chicago, Ill., ALR
PONTIUS, P. J.....1831 Chestnut St., Philadelphia, Pa., Op.
POOS, E. E. (1927).....Fisher Bldg., Detroit, Mich., OALR
POOS, GROVER HEWITT (1928)
University Club Bldg., St. Louis, Mo., OALR
POPE, E. C. (1930).....1931 1st Ave., Birmingham, Ala., ALR
PORTER, C. T. (1930)..520 Commonwealth Ave., Boston, Mass., ALR
*PORTER, E. H. (1912).....85 Madison St., Tiffin, O., OALR
PORTER, JESSE B. (1920).....415 S. 2nd St., Elkhart, Ind., OALR
PORTER, L. B. (1913).....199 Thayer St., Providence, R. I., OALR
*POST, LAWRENCE T. (1920)
Metropolitan Bldg., St. Louis, Mo., Op.
POST, M. HAYWARD (1918)
520 Metropolitan Bldg., St. Louis, Mo., Op.
POST, WINFRED B. (1921)
First National Bank Bldg., Carthage, Mo., OALR
*POSTLE, C. D. (1930).....240 E. State St., Columbus, O., ALR
*POTTER, ALLEN B. (1929), 3725 Kingshighway, St. Louis, Mo., ALR
POTTER, GEO. B. (1921).....Medical Arts Bldg., Omaha, Neb., OALR
POTTER, L. F.....68 Walnut St., New Bedford, Mass., OALR
POTTER, PETER (1918).....Motor Clinic Bldg., Butte, Mont., OALR
POTTER, W. A. (1930)
1355 David Whitney Bldg., Detroit, Mich., ALR
POTTER, W. W. (1927).....Medical Bldg., Knoxville, Tenn., OALR
POTTS, J. B.....Medical Arts Bldg., Omaha, Neb., ALR
*POWELL, LYLE STEPHENSON (1928)
813 Mass. St., Lawrence, Kan., OALR
*POWERS, EVERETT (1916)
Central Nat'l Bank Bldg., Carthage, Mo., OALR
PRANGEN, AVERY DE H. (1919), Mayo Clinic, Rochester, Minn., Op.
*PRATT, FRED J. (1912)
1753 Medical Arts Bldg., Minneapolis, Minn., OALR
PRATT, JOHN A. (1910)
1753 Medical Arts Bldg., Minneapolis, Minn., OALR
PRENDERGAST, D. A.....Medical Arts Bldg., Cleveland, O., OALR
PRESTON, H. G. (1930), Professional Bldg., Harrisonburg, Va., ALR
PRICE, JOHN B. (1929).....513 Swede St., Norristown, Pa., ALR

PRICE, NORMAN W. (1908), 445 3rd St., Niagara Falls, N. Y., OALR
PRINCE, ALEXANDER L. (1928), 36 Pearl St., Hartford, Conn., Op.
*PROETZ, A. W. (1923).....Beaumont Bldg., St. Louis, Mo., ALR
PROUD, WILLARD C. (1920)
Phys. and Surg. Bldg., St. Joseph, Mo., OALR
PROUT, A. W. (1920).....380 E. Town St., Columbus, O., OALR
PUTNAM, F. i.....501 St. Claire Bldg., San Jose, Cal., OALR
PYLE, WALLACE (1915)....15 Exchange Pl., Jersey City, N. J., OALR

Q

QUALLS, HARLEY WINFIELD (1928)
1531 Exchange Bldg., Memphis, Tenn., OALR
QUINBY, WM. O'G. (1919).....14 James St., Newark, N. J., OALR
QUINCY, J. E. (1927), 270 Commonwealth Ave., Boston, Mass., ALR
*QUITTNER, S. S.....3912 Prospect Ave., Cleveland, O., OALR

R

RADCLIFFE, MCCLUNEY, 1930 Chestnut St., Philadelphia, Pa., Op.
RALLS, C. T. (1916).....1st Nat'l Bank Bldg., Winfield, Kan., OALR
*RALSTON, W. WALLACE (1918)
1300 Walker Ave., Houston, Texas, Op.
RAMSEY, G. S. (1927), 1496 Mountain St., Montreal, Canada, Op.
RANDALL, HIRAM (1928).....81 Main St., Binghamton, N. Y., ALR
RAVDIN, B. D.....Central Union Bk. Bldg., Evansville, Ind., OALR
*RAVDIN, M.....Central Union Bk. Bldg., Evansville, Ind., OALR
RAY, VICTOR (1901).....19 W. 7th St., Cincinnati, O., Op.
REAVES, CHARLES R.
Reaves Infir., 117 W. Sycamore St., Greensboro, N. C., OALR
*REAVES, ROBERT GRISHAM (1928)
422 W. Cumberland Ave., Knoxville, Tenn., OALR
REAVES, W. P. (1913)
Reaves Infir., 117 W. Sycamore St., Greensboro, N. C., OALR
*RECTOR, A. E. (1915), 112 W. College Ave., Appleton, Wis., OALR
REED, WM. W. (1930).....Mills Bldg., Topeka, Kan., ALR
*REEDER, J. E. (1916).....Davidson Bldg., Sioux City, Ia., OALR
*REESE, ALGERNON B. (1928)
50 W. 52nd St., New York City, N. Y., Op.
REESE, MONROE D. (1926).....123 N. 8th St., Lebanon, Pa., OALR
REESE, WARREN S. (1921)..230 S. 21st St., Philadelphia, Pa., Op.
*REGER, H. S. (1908).....Physicians Bldg., Jamestown, N. Y., OALR
REICHE, O. C. (1916).....Markle Bk. Bldg., Hazleton, Pa., OALR
*REID, H. W. (1930).....19 Garfield Place, Cincinnati, O., Op.
REIM, HUGO.....417 Metropolitan Bldg., St. Louis, Mo., Op.
REINEKE, GEO. F. (1926).....Box 137, New Ulm, Minn., OALR
RENAUD, G. L.....510 Joseph Campan Ave., Detroit, Mich., OALR
RENNER, W. S. (1900).....341 Linwood Ave., Buffalo, N. Y., ALR

REPASS, R. E. (1920).....337 Lincoln Rd., Miami Beach, Fla., ALR
*REULING, F. H. (1924).....Black Bldg., Waterloo, Ia., OALR
*REYNOLDS, H. G. (1909), City Nat'l Bk. Bldg., Paducah, Ky., OALR
*REYNOLDS, J. S. (1918)
Medical Arts Bldg., Minneapolis, Minn., OALR
*RHODE, SOLON LAFAYETTE (1928)
238 N. 6th St., Reading, Pa., OALR
RICE, CLARENCE PRENTICE.....Breckenridge, Minn., OALR
RICE G. B. (1916).....99 Bay State Road, Boston, Mass., ALR
RICE, JOHN E. (1924).....390 Main St., Worcester, Mass., OALR
RICE, R. H. (1915).....331 Grove St., Milwaukee, Wis., OALR
*RICHARDS, L. G. (1924)..319 Longwood Ave., Boston, Mass., ALR
RICHARDSON, J. J. (1929)
Cook Hosp. and Beall Clinic, Fort Worth, Texas, ALR
RICHARDSON, S. A. (1925)
111 W. Adams St., Jacksonville, Fla., Op.
RICHIE, L. T. (1916).....McCormick Bldg., Trinidad, Colo., OALR
*RIDPATH, ROBERT F.
N. E. cor. 18th and Chestnut Sts., Philadelphia, Pa., LR
RIEGER, W. H. (1924).....Hanna Bldg., Cleveland, O., ALR
*RIGG, J. P. (1930)....Grand Island Clinic, Grand Island, Neb., ALR
RINDLAUB, ELIZABETH P.....de Lendrecie Bldg., Fargo, N. D., OALR
RINDLAUB, JOHN H.....First Trust Bldg., Pasadena, Cal., OALR
*RINGLE, CHAS. A. (1906).....Coronado Block, Greeley, Colo., ALR
RINGUEBERG, E. N. S. (1905), 13 Main St., Lockport, N. Y., Op.
RISDON, E. F. (1924).....30 Avenue Rd., Toronto, Canada, ALR
*RISSER, EDWARD D. (1928), 310 Choate Bldg., Winona, Minn., OALR
ROBB, MAYO (1929).....323 Ellis Bldg., Phoenix, Ariz., ALR
*ROBERTS, S. E. (1916)..Professional Bldg., Kansas City, Mo., ALR
ROBERTS, WM. H.....65 N. Madison Ave., Pasadena, Cal., Op.
*ROBERTSON, EDW. N. (1918)
132½ W. 6th St., Concordia, Kan., OALR
ROBERTSON, F. D.....Ashton Bldg., Grand Rapids, Mich., OALR
ROBINSON, H. T. (1916)....Medical Bldg., Cumberland, Md., OALR
*ROBINSON, R. E. (1920), 205½ E. Bremer Ave., Waverly, Ia., OALR
*ROBISON, J. T. (1930).....309 State St., New London, Conn., ALR
ROGERS, A. S.....105 S. Jefferson Ave., Saginaw, Mich., OALR
ROGERS, J. T.....Medical Arts Bldg., Montreal, Can., ALR
ROGERS, ROBERT MERRIAM (1928)
680 St. Johns Pl., Brooklyn, N. Y., Op.
ROLLER, LOUIS A. (1920)
446 Paris Ave., S. E., Grand Rapids, Mich., Retired
ROMMEL, JOHN C.....3701 N. Broad St., Philadelphia, Pa., Op.
*ROMONEK, PHILIP L. (1929)..Med. Arts Bldg., Omaha, Neb., OALR
*ROOKER, A. M. (1920).....Gluck Bldg., Niagara Falls, N. Y., ALR
ROOST, F. H. (1914).....Sioux City, Ia., OALR
ROSEBROUGH, F. H. (1929)....Moore Bldg., San Antonio, Tex., Op.
ROSENBAUM, J., 1396 St. Catherine's St. W., Montreal, Can., Op.
ROSENBAUM, MORRIS (1929), 604 E. 5th St., New York, N. Y., Op.

ROSENBERGER, H. C. (1929).....Rose Bldg., Cleveland, O., ALR
ROTH, ALBERT W. (1920), Medical Arts Bldg., Tulsa, Okla., OALR
*ROTHSCHILD, HAROLD J.....239 Lowry Bldg., St. Paul, Minn., OALR
ROTT, O. M. (1915)
Paulsen Medical and Dental Bldg., Spokane, Wash., OALR
ROW, GEO. S. (1911)
906 Hume-Mansur Bldg., Indianapolis, Ind., Op.
ROWLAND, JOHN F. (1911)
Medical Arts Bldg., Hot Springs, Ark., OALR
ROWLAND, W. D. (1923).....220 Clarendon St., Boston, Mass., Op.
*RUBENDALL, C. (1915).....Medical Arts Bldg., Omaha, Neb., OALR
RUBENSTEIN, A. H. (1930)..Med. Arts Bldg., Syracuse, N. Y., ALR
RUBY, F. M.....1940 E. 3rd Ave., Hibbing, Minn., OALR
RUCKER, C. W. (1929)..Med. Arts Bldg., Minneapolis, Minn., Op.
RUCKER, E. W., JR. (1916)
926 Woodward Bldg., Birmingham, Ala., ALR
RUDOLPHY, J. B. (1929).....1924 Pine St., Philadelphia, Pa., Op.
*RUEDEMANN, A. D. (1929)
Cleveland Clinic, Euclid Ave. and 93rd St., Cleveland, O., Op.
*RUNK, R. L. (1929)
Wash. Sanit. and Hosp., Takoma Pk., Washington, D. C., ALR
*RUSH, C. C.....907 U. S. Nat'l Bk. Bldg., Johnstown, Pa., OALR
RUSSELL, J. A. M. (1922).....813 Sassafras St., Erie, Pa., ALR
RUSSELL, R. E. (1924)..601 Pioneer Bk. Bldg., Waterloo, Ia., OALR
RUST, E. G.....Hanna Bldg., Cleveland, O., Op.
*RUTHERFORD, C. W. (1925)
University Hospital, Dept. Ophth., Iowa City, Iowa, Op.
RYAN, ED. R. (1930).....425 E. Water St., Milwaukee, Wis., Op.
RYAN, L. M. (1927).....Wall Bldg., St. Louis, Mo., ALR
RYCHENER, RALPH ORLANDO (1928)
1720 Exchange Bldg., Memphis, Tenn., Op.
RYDER, D. R.....151 Rock St., Fall River, Mass., ALR
*RYERSON, F. L. (1925).....Fine Arts Bldg., Detroit, Mich., OALR

S

SACCO, ANTHONY (1920)
440 New York Ave., Union City, N. J., OALR
*SALINGER, SAMUEL (1920)
25 E. Washington St., Chicago, Ill., ALR
SALLANDER, F. W. (1919)
385 New Orpheum Bldg., Sioux City, Iowa, OALR
*SAMUELS, BERNARD (1929), 57 W. 57th St., New York, N. Y., Op.
*SANDERS, JOHN GILLIS (1929)
Merchants Nat'l Bank Bldg., Mobile, Ala., ALR
*SANDERSON, DAVID D. (1916), 914 Stuart Bldg., Lincoln, Neb., OALR
*SANDERSON, H. H. (1906)
David Whitney Bldg., Detroit, Mich., OALR

SANOR, DANIEL GALBREATH, JR. (1928)
206 E. State St., Columbus, O., ALR
SARTAIN, PAUL J. (1921), 2006 Walnut St., Philadelphia, Pa., Op.
SAUER, W. E. (1904)....Beaumont Med. Bldg., St. Louis, Mo., ALR
*SAUER, W. W. (1910).....231 Third St., Marietta, O., OALR
SAVAGE, M. M. (1927).....1720 Eutaw Pl., Baltimore, Md., OALR
*SAVERY, C. E.....206 Sherland Bldg., South Bend, Ind., OALR
SCAL, JOSEPH COLEMAN (1926)
44 W. 74th St., New York City, N. Y., ALR
*SCALES, H. L. (1920).....Rorabaugh Bldg., Hutchinson, Kan., OALR
SCALES, J. L. (1915).....Medical Arts Bldg., Shreveport, La., OALR
SCALES, J. W. (1896).....204½ Main St., Pine Bluff, Ark., OALR
SCARLETT, HUNTER W. (1919)
230 S. 21st St., Philadelphia, Pa., Op.
*SCHALL, L. A. (1927), 270 Commonwealth Ave., Boston, Mass., ALR
SCHAPPERT, N. L. (1916)
59 S. Washington St., Wilkes-Barre, Pa., OALR
SCHENCK, C. P. (1920), Med. Arts Bldg., Fort Worth, Tex., OALR
SCHILD, E. H. (1906).....Brant Bldg., Canton, O., Op.
SCHILLER, A. N. (1918)
101 Central Park, West, New York City, N. Y., ALR
SCHLANSER, MAJOR A. E. (1921)
Walter Reed Gen. Hosp., Army Med. Center, Washington, D.C., OALR
SCHLEGEL, HERMAN T. (1916), 501 Third St., Wausau, Wis., OALR
*SCHLINDWEIN, G. WM.....138 W. 9th St., Erie, Pa., OALR
SCHLIVEK, KAUFMAN (1925)
1016 Fifth Ave., New York City, N. Y., Op.
*SCHLUER, E. P. (1930).....Metropolitan Bldg., St. Louis, Mo., ALR
SCHNEIDER, ANTON S. (1928), 56 Court St., Plattsburg, N. Y., Op.A
*SCHOLZ, R. P.....Metropolitan Bldg., St. Louis, Mo., ALR
*SCHRANT, J. H. (1916).....14 W. 1st St., Hutchinson, Kan., OALR
SCHULZE, E. C. (1926).....Med. Arts Bldg., Dallas, Tex., OALR
SCHURTER, M. A. (1924).....114 E. 7th St., Long Beach, Cal., OALR
*SCHUSTER, FRANK P. (1927)
2000 N. Mesa Ave., El Paso, Tex., OALR
SCHUSTER, S. A. (1925).....1st Nat'l Bk. Bldg., El Paso, Tex., ALR
SCHUTZ, W. H. (1921).....Bryant Bldg., Kansas City, Mo., OALR
SCHWARTZ, A. A. (1927)
941 Park Ave., New York City, N. Y., ALR
SCHWARTZ, FREDERICK O. (1919)
Metropolitan Bldg., St. Louis, Mo., Op.
SCHWARTZ, V. J. (1929), Med. Arts Bldg., Minneapolis, Minn., ALR
SCHWARTZ, WILLIAM A. (1928)
Goodrich Bldg., Phoenix, Ariz., OALR
SCHWEINITZ, GEO. E. DE (1905)
1705 Walnut St., Philadelphia, Pa., Op.
*SCOTT, MERLE E. (1926)....1st Nat'l Bk. Bldg., Massillon, O., OALR
*SCROGGS, J. J. (1921).....218 E. Third St., Beaver, Pa., Op.A
*SEARCY, H. B. (1912).....2501 6th St., Tuscaloosa, Ala., OALR

SEAY, D. E.....907 Mercantile Bk. Bldg., Dallas, Tex., OALR
*SEBERT, L. S. (1930).....Medical Arts Bldg., Toronto, Canada, Op.
*SECOY, F. L. (1922).....301 Davidson Bldg., Sioux City, Ia., OALR
SEDGWICK, O. W. (1917).....Ionia, Mich., OALR
SEDWICK, W. A.....Republic Bldg., Denver, Colo., Op.
*SEEGMAN, SIMON (1918).....Medical Bldg., Pittsburgh, Pa., ALR
SEELYE, W. K. (1920).....1215 4th Ave., Seattle, Wash., OALR
SELIGSTEIN, MILTON B. (1926)
801 Union and Planters Bk. Bldg., Memphis, Tenn., OALR
SENER, WALTER J.....Physicians Bldg., Philadelphia, Pa., Op.
SENSENEY, EUGENE TOWNER (1928)
316 Lister Bldg., St. Louis, Mo., ALR
SEWALL, E. C. (1910)
Stanford Univ. Hospital, San Francisco, Cal., ALR
*SEYDELL, E. M. (1916)
1030 First Nat'l Bank Bldg., Wichita, Kan., ALR
SHACKLETON, W. E. (1918)
2323 Prospect Ave., Cleveland, O., Op.
SHAFER, J. J. (1913).....Starks Bldg., Louisville, Ky., OALR
SHAHAN, W. E.....Metropolitan Bldg., St. Louis, Mo., Op.
*SHANKLIN, E. M.....575 Hohman St., Hammond, Ind., OALR
SHANNON, C. E. G.....1930 Chestnut St., Philadelphia, Pa., Op.
SHAPERO, I. M. (1919).....289 Alexander St., Rochester, N. Y., OALR
SHAPIRO, S. L. (1930).....25 E. Washington St., Chicago, Ill., ALR
SHARP, BENJ. S. (1926).....339 Thayer St., Providence, R. I., OALR
SHARRETT, GEO. O. (1921).....119 Bedford, Cumberland, Md., OALR
SHASTID, T. H. (1913).....Sellwood Bldg., Duluth, Minn., Op.
SHAWHAN, GLENN E. (1913)
Smallwood Bldg., Nampa, Idaho, OALR
*SHEA, J. J. (1919).....1018 Madison Ave., Memphis, Tenn., ALR
SHEAHAN, WM. L. (1926), 59 College St., New Haven, Conn., OALR
SHELLMAN, J. L. (1917)
Lowry Medical Arts Bldg., St. Paul, Minn., OALR
SHEMELEY, WM. G., JR., 1724 Spruce St., Philadelphia, Pa., OALR
SHERMAN, MAJ. A. ED., M. C., U. S. A. (1913)
Hdqts. U. S. Troops, Air Corp., Municipal Air Port,
Long Beach, Cal., OALR
SHERMAN, ELBERT S. (1912).....671 Broad St., Newark, N. J., Op.
*SHIELDS, J. M. (1915), 264 Metropolitan Bldg., Denver, Colo., Op.
SHIELDS, LAWRENCE.....Allen Bldg., Xenia, O., ALR
SHIRAS, HOWARD H. (1920)
624 Guardian Bldg., Cleveland, O., Op.
*SHIREY, O. M. (1925).....Osborn Bldg., Cleveland, O., ALR
SHOEMAKER, FERDINAND, U. S. Veterans' Hospital No. 67,
27th and Paseo, Kansas City, Mo., OALR
SHOEMAKER, S. A.....30 E. Church St., Orlando, Fla., OALR
SHOEMAKER, W. A. (1896)
1006 Carleton Bldg., St. Louis, Mo., Op.
SHREVE, OWEN M. (1905).....162 W. 8th St., Erie, Pa., Op.

SHULTS, E. J. G. (1920).....Haines Bldg., Eldorado, Kan., OALR
SHUMAN, G. H. (1917).....Park Bldg., Pittsburgh, Pa., Op.
SHUMATE, D. L.....500 Argyle Bldg., Kansas City, Mo., OALR
*SHURLY, BURT R.....62 Adams Ave., W., Detroit, Mich., ALR
SHUSTER, B. H. (1929)....1930 Chestnut St., Philadelphia, Pa., ALR
SHUTE, D. K.....1727 DeSales St., N.W., Washington, D. C., Op.
*SIDWELL, C. E. (1929).....Longmont Hosp., Longmont, Colo., ALR
*SIEGEL, FRANCIS X. (1922)
901 Union Central Life Bldg., Cincinnati, O., OALR
SILBERNAGEL, C. E. (1918)....240 E. State St., Columbus, O., OALR
SIMON, F. C. (1929).....914 Arcade Bldg., St. Louis, Mo., ALR
*SIMONDS, O. F.....Hanna Bldg., Cleveland, O., Op.
*SIMONS, N. S. (1930).....MacCornack Clinic, Whitehall, Wis., ALR
SIMPSON, H. LEE (1921)
1409 David Whitney Bldg., Detroit, Mich., ALR
SIMPSON, JOHN REID.....825 Highland Bldg., Pittsburgh, Pa., ALR
SIMPSON, M. B. (1930).....Rialto Bldg., Kansas City, Mo., ALR
*SIMPSON, W. LIKELY (1911)
Phys. and Surg. Bldg., Memphis, Tenn., ALR
SINAMARK, ANDREW (1928), 205 Power Bldg., Hibbing, Minn., OALR
SINGLETON, E. M. (1897)
111 E. Church, St., Marshalltown, Ia., R
SISSON, RAYMOND J. (1928)
753 David Whitney Bldg., Detroit, Mich., Op.
*SKILLERN, S. R., JR. (1925), 1734 Pine St., Philadelphia, Pa., ALR
SKIRBALL, JOSEPH J. (1927)
353 Commonwealth Ave., Boston, Mass., OALR
*SLEIGHT, R. D. (1906)....303 Post Bldg., Battle Creek, Mich., Op.
*SLOAN, HENRY L. (1919).....6 W. 7th St., Charlotte, N. C., Op.
SLOANE, H. O. (1930).....1717 Pine St., Philadelphia, Pa., Op.
*SLOCUM, GEORGE (1906)
First Nat'l Bank Bldg., Ann Arbor, Mich., Op.
SMIT, WM. MAURICE (1928)
719 Missouri Theater Bldg., St. Louis, Mo., ALR
SMITH, ARTHUR E.....Donaldson Bldg., Minneapolis, Minn., OALR
*SMITH, CARLIE SOUTER (1927)
214 N. Jefferson Ave., Springfield, Mo., OALR
*SMITH, CARROLL L. (1929)
Paulsen Medical and Dental Bldg., Spokane, Wash., OALR
*SMITH, DORLAND (1910), 836 Myrtle Ave., Bridgeport, Conn., Op.A
*SMITH, D. S. (1917).....State Bank Bldg., La Crosse, Wis., OALR
SMITH, E. TERRY.....36 Pearl St., Hartford, Conn., Op.
*SMITH, GEORGE B. (1914).....Harbin Hospital, Rome, Ga., OALR
SMITH, H. A. (1925)....315 E. Philadelphia St., Whittier, Cal., OALR
SMITH, J. M.....134½ N. Patterson St., Valdosta, Ga., OALR
SMITH, R. C.....Board of Trade Bldg., Superior, Wis., OALR
*SMITH, R. N. (1929).....Medical Arts Bldg., Tulsa, Okla., ALR
SMITH, STANLEY.....121 University Pl., Pittsburgh, Pa., Op.

SMITH, VICTOR C. (1912)
1707 Pere Marquette Bldg., New Orleans, La., Op.
*SMITH, WM. L. (1930)
Medical Arts Bldg., Salt Lake City, Utah, Op.
*SNAPP, CARL FOSTER (1928)
604 Medical Arts Bldg., Grand Rapids, Mich., ALR
*SNEED, CARL M.....506 W. Broadway, Columbia, Mo., OALR
SNELL, ALBERT C. (1925)
53 S. Fitzhugh St., Rochester, N. Y., Op.
*SNYDER, W. H.....2001 Callingwood Ave., Toledo, O., OALR
SOMBERG, J. S. (1923).....470 Park Ave., New York, N. Y., Op.
*SONNENSCHEIN, R. (1915)
180 N. Michigan Ave., Chicago, Ill., ALR
*SPAETH, EDMUND B. (1928)
1930 Chestnut St., Philadelphia, Pa., Op.
*SPAKE, LAVERNE B. (1920)
322 Brotherhood Bldg., Kansas City, Kan., ALR
SPALDING, J. A. (1896).....627 Congress St., Portland, Me., Op.A
*SPENCER, FRANK R. (1904), Physicians Bldg., Boulder, Colo., OALR
SPENCER, GEORGE ALBERT (1928)
1127 Eleventh St., Sacramento, Cal., OALR
SPENCER, S. C. (1929).....605 Columbus Ave., Waco, Texas, Op.
SPENCER, W. H.....1830 S. Rittenhouse Sq., Philadelphia, Pa., ALR
SPENGLER, JOHN ARTHUR (1916)
423 S. Main St., Geneva, N. Y., Op.
*SPIESMAN, I. G. (1929)....25 E. Washington St., Chicago, Ill., ALR
SPITZE, E. C. (1916).....Murphy Bldg., East St. Louis, Ill., Op.
*SPROULE, RALPH P. (1928)
141 E. Wisconsin Ave., Milwaukee, Wis., OALR
ST. CLAIR, C. T. (1930)
Corner Bland and Ramsey Sts., Bluefield, W. Va., ALR
STAHLMAN, FRED C.....Commercial Bldg., Waynesburg, Pa., OALR
STANBERRY, HENRY.....556 Doctors Bldg., Cincinnati, O., Op.
*STANFORD, J. B.....1052 Madison Ave., Memphis, Tenn., OALR
*STANLEY, C. R. (1930).....Worthington, Minn., ALR
*STANTON, HARMON LAMONTTE (1928)
Cleveland Life Bldg., Evansville, Ind., OALR
STARK, JESSE B.(1925), 45 Park Ave., New York City, N. Y., Op.
STARK, WM. B. (1927).....Mayo Clinic, Rochester, Minn., ALR
*STAUFFER, FRED (1912)
Medical Arts Bldg., Salt Lake City, Utah, OALR
*STEELE, WILLARD (1928)
Provident Bldg., Chattanooga, Tenn., OALR
STEIM, J. M. (1914)
First Nat'l Bank Bldg., New Kensington, Pa., Op.
STEINFELD, A. L. (1922).....125 15th St., Toledo, O., OALR
*STEVENSON, D. W.....165 E. Market St., Akron, O., OALR
*STEVENSON, WALTER (1915).....W. C. U. Bldg., Quincy, Ill., OALR

*Stewart, Walter E. (1920)
Tribune Bldg., Terre Haute, Ind., OALR
Stieren, E. (1905)....626 Union Trust Bldg., Pittsburgh, Pa., Op.
Stiles, Chas. M. (1908)....1130 Spruce St., Philadelphia, Pa., Op.
Stillson, H.....Seabord Bldg., Seattle, Wash., OALR
Stimson, George W. (1919).....Packard Bldg., Warren, O., OALR
Stinson, Wm. David (1928)
805 Medical Arts Bldg., Memphis, Tenn., ALR
Stitt, H. L. (1927).....656 Doctors Bldg., Cincinnati, O., OALR
Stockler, J. A. (1917).....332 North Fifth St., Reading, Pa., OALR
Stokes, Wm. H. (1926).....4105 Live Oak St., Dallas, Tex., Op.
*Stonecypher, D. D.....109 S. 9th St., Nebraska City, Neb., ALR
Stone, H. B. (1930).....Medical Arts Bldg., Roanoke, Va., ALR
*Stotter, Arthur L. (1916), 1148 Euclid Ave., Cleveland, O., OALR
Stout, Philip S.....269 S. 19th St., Philadelphia, Pa., ALR
Stoutenborough, Wm. A. (1927)
21 East State St., Columbus, O., OALR
Strader, Geo. L. (1904)..408 Hynds Bldg., Cheyenne, Wyo., OALR
*Strauss, J. F. (1925).....104 S. Michigan Blvd., Chicago, Ill., ALR
Strickler, D. A.....Republic Bldg., Denver, Colo., Op.
Strong, J. C. (1915).....810 Harrison Ave., Leadville, Colo., OALR
Stroud, Edward Frank (1926)
Medical Professional Bldg., Corpus Christi, Tex., ALR
Strout, E. S. (1907).....74 S. 11th St., Minneapolis, Minn., OALR
Strout, G. E.(1921), 1009 Nicollet Ave., Minneapolis, Minn., OALR
*Stucky, J. A.(1896), Fayette Nat'l Bk. Bldg., Lexington, Ky., OALR
*Stueber, F. G. (1905).....209 W. North St., Lima, O., OALR
*Stueber, Paul J. (1917).....209 W. North St., Lima, O., OALR
Sturges, Leigh F. (1920)
121 E. 60th St., New York City, N. Y., ALR
Sturm, S. A. (1916)
7036 Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
*Suker, Geo. F. (1896).....25 E. Washington St., Chicago, Ill., Op.
Sullivan, J. J., Jr. (1923)
228 Scranton Life Bldg., Scranton, Pa., ALR
Sulzer, G. A. (1907).....327 E. State St., Columbus, O., OALR
*Sulzman, Frank Malcolm (1922)
1831 Fifth Ave., Troy, N. Y., OALR
Summers, Wm. S. (1927)
1613 David Whitney Bldg., Detroit, Mich., OALR
Sutherland, J. M. (1929)
8-257 General Motors Bldg., Detroit, Mich., ALR
Swab, Charles Marion (1928)
Medical Arts Bldg., Omaha, Neb., Op.
Swan, C. J. (1911).....636 Church St., Evanston, Ill., OALR
Sweet, Robert B. (1926)
Pacific S. W. Bk. Bldg., Long Beach, Cal., OALR
*Swerdfeger, E. B.(1927), 1008 Republic Bldg., Denver, Colo., ALR
Swetmam, C. K. (1915).....Masonic Temple, Prescott, Ariz., ALR

T

TANGEMAN, H. F. (1919)
606 Union Central Bldg., Cincinnati, O., ALR
*TAQUINO, GEORGE J. (1920)
208 Audubon Bldg., New Orleans, La., ALR
TARUN, WILLIAM.....104 West Madison St., Baltimore, Md., OALR
TASSMAN, ISAAC C. (1929)
Medical Arts Bldg., Philadelphia, Pa., Op.
*TAYLOR, CHARLES B. (1921).....118 E. 2nd St., Ottumwa, Ia., ALR
TAYLOR, LILLIAN E. (1905)
25 E. Washington St., Chicago, Ill., ALR
TAYLOR, M. C. (1915).....1204 May Bldg., Pittsburgh, Pa., Op.
*TEALL, RAYMOND EDWIN (1928)
Professional Bldg., Kansas City, Mo., ALR
TEBBUTT, H. K. (1930).....240 State St., Albany, N. Y., ALR
*THEOBALD, GEORGIANA DVORAK (1929)
715 Lake St., Oak Park, Ill., Op.
*THEOBALD, JOHN J. (1929)
58 E. Washington St., Chicago, Ill., ALR
*THEOBALD, W. H. (1930)..307 N. Michigan Ave., Chicago, Ill., ALR
THIGPEN, C. A.....401 S. Court St., Montgomery, Ala., OALR
THOMAS, C. D. (1914), Central Nat'l Bank Bldg., Peoria, Ill., OALR
*THOMAS, FRANCIS W. (1916), 327 E. State St., Columbus, O., OALR
THOMAS, H. G. (1926).....411 30th St., Oakland, Cal., OALR
*THOMAS, JAMES W. (1928).....Sharp Bldg., Lincoln, Neb., OALR
THOMASHEFSKY, MILTON (1930)
135 Eastern Parkway, Brooklyn, N. Y., ALR
THOMASON, H. E.....Rialto Bldg., Kansas City, Mo., ALR
THOMASSON, W. J. (1910).....942 York St., Newport, Ky., ALR
THOMPSON, E. H.....8 W. 9th St., Cincinnati, O., OALR
THOMPSON, G. H. (1909).....74 North St., Pittsfield, Mass., OALR
THOMPSON, HOWARD E.(1928), 1100 Main St., Dubuque, Ia., OALR
THOMPSON, PETER H. (1914)
308 Commonwealth Ave., Boston, Mass., Op.
THOMSON, J. J. (1907)
531 East Lincoln Ave., Mt. Vernon, N. Y., ALR
*TILDERQUIST, D. L. (1925), 425 Bradley Bldg., Duluth, Minn., OALR
TILLOTSON, R. S. (1930)....Woodland Clinic, Woodland, Cal., ALR
TIMBERMAN, ANDREW.....Beggs Bldg., Columbus, O., OALR
TINDOLPH, L. W.....1st Nat'l Bank Bldg., Olean, N. Y., ALR
TINGLEY, LOUISA PAINE (1913)
9 Massachusetts Ave., Boston, Mass., Op.
Also 171 Westminster St., Providence, R. I.
TITSWORTH, GUY.....111 W. 4th St., Sedalia, Mo., OALR
*TIVNEN, RICHARD J.....30 N. Michigan Ave., Chicago, Ill., OALR
*TOBEY, GEO. L., JR.....270 Commonwealth Ave., Boston, Mass., ALR
*TODD, GROVER CLEVELAND (1922)
6133 Jenkins Arcade Bldg., Pittsburgh, Pa., ALR

*Tomassene, R. A. (1922)
Wheeling Bank and Trust Bldg., Wheeling, W. Va., OALR
Tomb, E. H.....110 E. Wisconsin Ave., Milwaukee, Wis., ALR
Tomlin, W. S. (1912)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
Toms, S. W. S. (1909).....120 S. Broadway, Nyack, N. Y., OALR
*Tooker, C. W. (1907).....Carleton Bldg., St. Louis, Mo., Op.
*Toot, J. F. (1930).....First Nat'l Bank, Canton, O., Op.
Townsend, John Ferrars (1923)
85 Wentworth St., Charleston, S. C., OALR
Trible, G. B. (1920)
1801 Eye St., N. W., Washington, D. C., OALR
*Trimble, Clarence S. (1920)
405 Citizens Nat'l Bank Bldg., Emporia, Kan., OALR
Tripp, Ira A. (1920).....436 Rose Bldg., Cleveland, O., OALR
Tripp, Leroy R.....Security Bldg., Sioux City, Ia., OALR
*Tucker, Gabriel (1923)....326 S. 19th St., Philadelphia, Pa., B
*Tucker, James C. (1920).....Arcade Bldg., Beatrice, Neb., OALR
Tuckerman, W. C. (1911)..733 Osborn Bldg., Cleveland, O., Op.
*Tuckerman, W. H.....733 Osborn Bldg., Cleveland, O., OALR
Turnbull, F. M. (1916)
806 Pacific Mutual Bldg., Los Angeles, Cal., ALR
*Turner, H. H. (1914), 902 Professional Bldg., Pittsburgh, Pa., Op.
Turner, Oliver W. (1909), Masonic Temple, Augusta, Me., OALR

U

Uhler, S. Mann (1930).....104 N. 8th St., Allentown, Pa., ALR
Upham, Helen F. (1907), 305 3rd Ave., Asbury Park, N. J., OALR
*Uren, C. T. (1919), 537 City Nat'l Bank Bldg., Omaha, Neb., ALR
Urner, M. H.....2600 Union Central Bldg., Cincinnati, O., OALR

V

Vail, H. H. (1924).....24 E. 8th St., Cincinnati, O., ALR
Van Dellan, A. L. (1930)..6857 S. Halsted St., Chicago, Ill., Op.
Van Kirk, Frank J. (1920)
Bellingham Nat'l Bank Bldg., Bellingham, Wash., OALR
*Van Horn, A. F. (1930)....514 Central Ave., Plainfield, N. J., ALR
Van Meter, Eugene R. (1927)
4473 Washington Ave., St. Louis, Mo., ALR
Van Poole, G. McD. (1925)
Young Bldg., Honolulu, T. Hawaii, OALR
Vaughan, G. E.....711 3rd St., Louisville, Ky., OALR
Vaughn, E. H.....Citizens Nat'l Bank, Tyler, Tex., OALR
Veasey, C. A., Sr. (1905)
Paulsen Medical and Dental Bldg., Spokane, Wash., OALR

VEASSY, C. A., JR. (1927)
404 Paulsen Medical and Dental Bldg., Spokane, Wash., OALR
VINSONHALER, F. (1902)....Urquhart Bldg., Little Rock, Ark., OALR
*VOIGT, C. B. (1912).....1702 Broadway, Mattoon, Ill., OALR
*VON COLDITZ, G. THOMSEN
25 E. Washington St., Chicago, Ill., OALR
*VON DER HEYDT, ROBERT (1926)
25 E. Washington St., Chicago, Ill., Op.

W

*WAGENER, HENRY P. (1926)....Mayo Clinic, Rochester, Minn., Op.
WAGERS, ARTHUR J. (1920)
1429 Spruce St., Philadelphia, Pa., ALR
*WAGNER, CARL B. (1912).....30 North State St., Chicago, Ill., OALR
WAGNER, W. A. (1927)
914 American Bank Bldg., New Orleans, La., ALR
*WALKER, CHAS. C. (1920), 604 Maccabee Bldg., Detroit, Mich., Op.
WALKER, C. E. (1920).....517 Jacobson Bldg., Denver, Colo., Op.
*WALKER, GEO. H. (1927).....172 Main St., Winona, Minn., OALR
WALKER, HERBERT S.....30 N. Michigan Ave., Chicago, Ill., OALR
WALKER, WALDO WEBSTER (1928)
99 Bay State Road, Boston, Mass., ALR
WALSH, JOSEPH MARK (1922).....Rapid City, S. D., OALR
WARD, J. W. (1930)..Exchange Bank Bldg., Greenville, Tex., ALR
*WARNER, ROBT. J. (1929).....Doctors Bldg., Nashville, Tenn., Op.
*WARREN, WILLIAM C., JR. (1928)
618 Atlanta Nat'l Bank Bldg., Atlanta, Ga., ALR
WARWICK, HAROLD LYNWOOD (1928)
913 Medical Arts Bldg., Fort Worth, Texas, ALR
WATKINS, EDWIN DIAL (1926)
1412 Exchange Bldg., Memphis, Tenn., OALR
*WATKINS, H. R. (1930)....Griesheim Bldg., Bloomington, Ill., ALR
WATSON, H. D. (1915).....151 Front St., Binghamton, N. Y., OALR
WATSON, JAMES A.....74 S. 11th St., Minneapolis, Minn., OALR
*WATSON, R. S. (1923)..120 N. Michigan St., Saginaw, Mich., OALR
*WAUGH, J. M.
Cleveland Clinic, Euclid Ave. at 93rd St., Cleveland, O., OALR
*WEAVER, JOHN O. (1929)
608½ W. Sheridan Ave., Shenandoah, Ia., ALR
WEAVER, T. W. (1914).....201½ N. Main St., Wichita, Kan., Op.
*WEBER, HENRY CHARLES (1928)
U. S. Naval Hospital, Chicago, Ill., OALR
WEBSTER, F. R. (1930)
720 State Tower Bldg., Syracuse, N. Y., Op.
*WEED, HARRY M. (1908)....196 Linwood Ave., Buffalo, N. Y., Op.
WEEKS, JOHN E....360 Edgecliff Road, Portland, Ore., Retired, Op.

*Weeks, Webb W. (1923)
20 E. 53rd St., New York City, N. Y., Op.
*Weeth, C. R. (1930).....37 Remsen St., Brooklyn, N. Y., ALR
Weidlein, Ivan F. (1930).....Osborn Bldg., Cleveland, O., ALR
*Weih, E. P. (1920).....Wilson Bldg., Clinton, Ia., OALR
Weil, A. I.....3503 Prytania St., New Orleans, La., ALR
*Weinberger, Nelson S. (1928)
Robert Packer Hospital, Savre, Pa., OALR
Weinstein, M. A. (1927), 1309 Spruce St., Philadelphia, Pa., ALR
Weisser, E. A.....806 May Bldg., Pittsburgh, Pa., Op.
Welch, Robert S. G. (1921)
46 State Circle, Annapolis, Md., OALR
*Wells, David W. (1908), Hotel Westminster, Boston, Mass., Op.
Wells, George S.....8 E. Arrellaga, Santa Barbara, Cal., ALR
Welty, C. F. (1926).....210 Post St., San Francisco, Cal., ALR
*Wencke, Carl G.....Battle Creek Sanit., Battle Creek, Mich., ALR
*Wendel, J. S. (1922)
744 David Whitney Bldg., Detroit, Mich., ALR
*Wenner, W. T. (1930).....St. Mary's Bldg., St. Cloud, Minn., Op.
Wessels, Andrew B. (1929)
Medical-Dental Bldg., San Diego, Cal., ALR
Westlake, Samuel B., Beaumont Med. Bldg., St. Louis, Mo., ALR
Wetherbee, Charles (1926)
Booth Bldg., Twin Falls, Idaho, OALR
Wetzel, John O. (1930)
Capitol Saving and Loan Bldg., Lansing, Mich., Op.
Wexler, David (1930).....55 E. 86th St., New York, N. Y., Op.
*Weymann, M. F. (1929)
2007 Wilshire Blvd., Los Angeles, Cal., Op.
Whalen, Edward Joseph (1928)
750 Main St., Hartford, Conn., ALR
Wheeler, Ernest C.....Townsend Bldg., Tacoma, Wash., OALR
*Wheeler, J. M. (1920).....30 W. 59th St., New York, N. Y., Op.
Wheeler, Merritt W.....1027 Lowry Bldg., St. Paul, Minn., OALR
Whelan, G. L. (1925).....2100 Walnut St., Philadelphia, Pa., ALR
*Wherry, W. P. (1910), 1500 Med. Arts Bldg., Omaha, Neb., OALR
Whisnant, A. M.....517 Professional Bldg., Charlotte, N. C., OALR
Whitaker, J. (1917).....905 Congress Bldg., Miami, Fla., OALR
White, Chas. P.....507 Citizens Bk. Bldg., Wilmington, Del., OALR
White, E. Hamilton (1912)
1610 Sherbrooke St., W., Montreal, Canada, ALR
White, O. E. (1930).....28 N. Main St., Cortland, N. Y., ALR
White, William Beverly (1928)
813 Gurley Bldg., Stamford, Conn., OALR
*Whitney, E. L. (1930)..Henry Ford Hospital, Detroit, Mich., ALR
Wiener, Alfred (1909), 550 Park Ave., New York, N. Y., OALR
*Wiener, Meyer.....900 Carleton Bldg., St. Louis, Mo., Op.
Wiggin, Wm. I. (1929).....202 Central St., Lowell, Mass., ALR

WILBUR, EDWARD P.
Bank of Kalamazoo Bldg., Kalamazoo, Mich., OALR
*WILDER, WM. H. (1896), 122 S. Michigan Blvd., Chicago, Ill., Op.
WILKINSON, A. P. (1930).....Fisher Bldg., Detroit, Mich., ALR
WILKINSON, OSCAR (1930)
1408 L St., N. W., Washington, D. C., OALR
*WILLIAMS, HORACE J. (1922)
5908 Green St., Germantown, Pa., ALR
WILLIAMS, M. M. (1927), Medical Arts Bldg., Scranton, Pa., Op.
*WILLIAMS, W. C. (1906).....Jefferson Bldg., Peoria, Ill., OALR
*WILLIAMS, WALTON H. (1921), Castell Bldg., Middletown, O., OALR
WILLSON, WESLEY W. (1928)
2204 Eaton Tower, Detroit, Mich., OALR
*WINDHAM, ROBERT EDWARD (1928)
234 W. Beauregard Blvd., San Angelo, Texas, OALR
WINGER, IRA B. (1927).....225 Michigan St., Toledo, O., OALR
*WINTER, GEORGE E. (1921).....Sun Bldg., Jackson, Mich., OALR
*WINTER, JOHN A. (1923).....205 W. 2nd St., Duluth, Minn. OALR
*WISE, RALPH C. (1914).....News Bldg., Mansfield, O., OALR
WOLD, K. C. (1924)
1027 Lowry Med. Arts Bldg., St. Paul, Minn., OALR
*WOLF, SAMUEL (1929)....55 E. Washington St., Chicago, Ill., Op.
*WOLFE, CLAUDE T. (1916)....Heyburn Bldg., Louisville, Ky., OALR
*WOLFE, OTIS R. (1919)
415 Masonic Temple, Marshalltown, Ia., OALR
WOOD, DOUGLAS R.....Medical Arts Bldg., Minneapolis, Minn., OALR
WOOD, EARL LEROY (1928), 192 Roseville Ave., Newark, N. J., ALR
WOOD, V. VISSCHER (1928)
201 Beaumont Medical Bldg., St. Louis, Mo., ALR
WOODRUFF, FREDERICK E., Metropolitan Bldg., St. Louis, Mo., Op.
*WOODRUFF, H. W.....500 N. Ottawa St., Joliet, Ill., OALR
WOODRUFF, T. A.....309 State St., New London, Conn., Op.
WOODRUFF, WALTER STUART (1927)
421 Huguenot St., New Rochelle, N. Y., ALR
*WOODS, R. H.....Neustadt Bldg., La Salle, Ill., Op.
WOODSON, BURBANK PALMER (1928)
Woodson Eye, Ear, Nose and Throat Hosp., Temple, Tex., OALR
WOODWARD, F. D. (1929).....Box 162, University, Va., ALR
*WOODWORTH, WM. P. (1927)
1424 Maccabee Bldg., Detroit, Mich., OALR
WORMLEY, H. R.....Talcott Bldg., Rockford, Ill., OALR
WRIGHT, C. DARCY (1921)
Medical Arts Bldg., Minneapolis, Minn., OALR
WRIGHT, J. B.....Professional Bldg., Raleigh, N. C., ALR
WRIGHT, J. W. M.....Hume-Mansur Bldg., Indianapolis, Ind., ALR
WRIGHT, WALTER WALKER (1926)
Medical Arts Bldg., Toronto, Canada, Op.

WURDEMANN, HARRY V.....320 Stimson Bldg., Seattle, Wash., Op.
*WURTZ, W. J. M. (1913).....333 Linwood Ave., Buffalo, N. Y., ALR
WYLIE, C. B.....269 High St., Morgantown, W. Va., OALR

Y

YANKAUER, SIDNEY, 121 E. 60th St., New York City, N. Y., ALR
YAZEL, HERMAN E.....733 Rialto Bldg., Kansas City, Mo., OALR
DE YOANNA, A. A.....111 Pierrepont St., Brooklyn, N. Y., ALR
*YOST, WALTER MILTON (1928)
Peoples Bank Bldg., Rochester, Pa., Op.
Also Westinghouse Bldg., Pittsburgh, Pa.
YOUNG, CHARLES A. (1926)
Medical Arts Bldg., Roanoke, Va., Op.

Z

ZEHNDER, A. CHARLES (1919)
188 Roseville Ave., Newark, N. J., Op.
ZEMER, S. G.....950 Stuart Bldg., Lincoln, Neb., OALR
ZENTMAYER, WILLIAM
Suite 803, S.E. cor. 20th & Chestnut Sts., Philadelphia, Pa., Op.
*ZIEGELMAN, ED. F. (1929)..450 Sutter St., San Francisco, Cal., ALR
ZINKHAN, A. M. (1920), The Rochambeau, Washington, D. C., ALR
*ZUBAK, MATTHEW FRANCIS
Citizens Mutual Trust Bldg., Wheeling, W. Va., OALR

GEOGRAPHICAL LIST

ALABAMA

BIRMINGHAM

HARRISON, W. G. ..... Empire Bldg., OALR
KINCANNON, LEROY T. ..... The Woodward, OALR
MAUMENEE, A. E. ..... 711 Empire Bldg., OALR
POPE, ERNEST C. ..... 1931 1st Ave., ALR
RUCKER, E. W., JR. ..... 926 Woodward Bldg., ALR

MOBILE

SANDERS, JOHN GILLIS ..... Merchants Nat'l Bank Bldg., ALR

MONTGOMERY

MERTINS, PAUL S. ..... 1115 Bell Bldg., OALR
THIGPEN, C. A. ..... 401 S. Court St., OALR

SELMA

KIRKPATRICK, SAMUEL ..... 902 Alabama Ave., OALR

TUSCALOOSA

SEARCY, H. B. ..... 2501 Sixth St., OALR

ARIZONA

MESA

MCCARTHY, OLIVER P. ..... Valley Bank Bldg., OALR

PHOENIX

MCLOONE, JOHN J. ..... 611 Heard Bldg., OALR
MOSS, H. E. ..... Luhrs Bldg., OALR
ROBB, MAYO ..... 323 Ellis Bldg., ALR
SCHWARTZ, WM. A. ..... 605 Goodrich Bldg., OALR

PRESCOTT

SWETNAM, C. K. ..... Masonic Temple, ALR

ARKANSAS

FORT SMITH

BUCKLEY, J. H. ..... 1st National Bank Bldg., OALR
DORENTE, D. R. ..... 1st National Bank Bldg., OALR
MOULTON, EVERETT C. ..... Merchants National Bank Bldg., OALR
MOULTON, HERBERT ..... Merchants National Bank Bldg., OALR

HOT SPRINGS

KING, O. H. ..... Medical Arts Bldg., OALR
ROWLAND, JOHN F. ..... Medical Arts Bldg., OALR

LITTLE ROCK

CALDWELL, ROBERT.....831 New Donaghey Bldg., OALR
MAHONEY, PAUL LEO.....Donaghey Bldg., ALR
MOORE, ROBERT B.....Trinity Hospital, ALR
VINSONHALER, F.....Urquhart Bldg., OALR

PINE BLUFF

SCALES, J. W.....204½ Main St., OALR

ROGERS

KOOBS, H. J. G.....117½ W. Walnut St., OALR

CALIFORNIA

ALTADENA

LAFORCE, B. D.....1140 Beverly Way, OALR

ANAHEIM

NEIGHBORS, CLARENCE A.....120 S. Los Angeles St., OALR

BAKERSFIELD

PACKARD, LOUIS A.....Haberfelde Bldg., OALR

HOLLYWOOD

LEMERE, H. B.....607 Hollywood Security Bldg., OALR

LA JOLLA

HESS, WM. L.....7478 Eads Ave., OALR

LONG BEACH

SCHURTER, M. A.....114 E. 7th St., OALR
SHERMAN, MAJ. A. ED.....M. C., U.S.A., Hdqts. U. S. Troops, Air Corps, Municipal Air Port, OALR
SWEET, ROBERT B.....Pacific S. W. Bank Bldg., OALR

LOS ANGELES

BELLIN, JOSEPH.....523 W. 6th St., OALR
BOURBON, OLIVER P.....520 W. 7th St., OALR
BROWN, J. M.....1136 W. 6th St., ALR
CRANE, WALTER RAYMOND.....756 S. Broadway, ALR
DAYTON, G. O.....1401 S. Hope St., OALR
DETLING, FRANK E.....500 S. Lucas St., OALR
FISHER, CARL.....1930 Wilshire Blvd., Op.
GOSNEY, CHARLES W.....Medical Arts Bldg., Op.
HOPKINS, W. E.....321 S. Lorraine Blvd., OALR
IRVINE, A. R.....1144 Roosevelt Bldg., Op.
KEIPER, GEORGE F., JR.....727 W. 7th St., ALR
KIEFER, HUGO A.....Brockman Bldg., OALR
KRESS, GEORGE H.....245 Bradbury Bldg., OALR
LEFLER, ANNA B.....610 S. Broadway, OALR
LEWIS, EUGENE R.....Roosevelt Bldg., ALR

MAGHY, CHARLES A.....Wilshire Medical Bldg., Op.
TURNBULL, F. M.....806 Pacific Mut. Bldg., ALR
WEYMANN, MORIE FREDERICK.....2007 Wilshire Blvd., Op.

OAKLAND

BEAUDOUX, HENRY ALEXANDER.....230 Grand Ave., OALR
O'CONNOR, RODERIC P.....1904 Franklin St., Op.
THOMAS, H. G.....411 30th St., OALR

PALOS VERDES ESTATES

STEIN, O. J.....ALR

PASADENA

CADY, D. W.....65 N. Madison Ave., Op.
HEITGER, J. D.....First Trust Bldg., OALR
HENNINGER, L. L.....Professional Bldg., OALR
RINDLAUB, JOHN H.....First Trust Bldg., OALR
ROBERTS, WM. H.....65 N. Madison Ave., Op.

SACRAMENTO

BRIGGS, W. E.....1027 10th St., OALR
SPENCER, GEORGE ALBERT.....1127 11th St., OALR

SAN DIEGO

BROWN, CHARLES WILLIAM.....Medico-Dental Bldg., ALR
BURTON, FRANK A.....Medico-Dental Bldg., ALR
DURR, S. A.....Medico-Dental Bldg., Op.
HIGBEE, D. R.....3245 Fourth St., ALR
WESSELS, ANDREW BENJAMIN.....Medico-Dental Bldg., ALR

SAN FRANCISCO

BARKAN, ADOLPH.....490 Post St., OALR
BARKAN, HANS.....490 Post St., Op.
BARKAN, OTTO.....490 Post St., Op.
BLAKE, WM. F.....490 Post St., Op.
BROWN, H. ALEXANDER.....291 Geary St., Op.
DERIVER, J. P.....Veterans Bureau, OALR
DOLMAN, PERCIVAL.....490 Post St., Op.
GRAHAM, H. B.....490 Post St., ALR
GREEN, A. S.....The Green's Eye Hospital, Op.
GREEN, L. D.....The Green's Eye Hospital, Op.
HEBERT, ARTHUR W.....450 Sutter St., OALR
IRVINE, ROBERT STEELE.....490 Post St., ALR
MCCOOL, J. L.....450 Sutter St., Op.
SELFRIDGE, G. L.....384 Post St., ALR
SEWALL, E. C.....Stanford Univ. Hospital, ALR
WELTY, C. F.....210 Post St., ALR
ZIEGELMAN, EDWARD FRANK.....450 Sutter St., ALR

SAN JOSE

PUTNAM, F. I.....501 St. Claire Bldg., OALR

SANTA BARBARA

JEAN, G. W.....7 E. Valerio, Op.
JOHNSTON, WM. H.....1421 Chapala St., OALR
LEWIS, J. D.....1534 State St., OALR
MELLINGER, WM. J.....120 E. Sola St., ALR
WELLS, GEORGE S.....8 E. Arrellaga, ALR

SANTA MARIA

BAIRD, CHAS. G.....301 E. Chapel St., OALR

SANTA MONICA

LEVENGOOD, H. W.....327 Wilshire Blvd., OALR

WHITTIER

SMITH, H. A.....315 E. Philadelphia St., OALR

WOODLAND

TILLOTSON, R. S.....Woodland Clinic, ALR

COLORADO

ALAMOSA

HERRIMAN, L. L.....OALR

BOULDER

FISCHER, V. B.....203 Mercantile Bank Bldg., OALR
SPENCER, FRANK R.....Physicians Bldg., OALR

COLORADO SPRINGS

CHAPMAN, S. JEFFERSON.....402 Burns Bldg., ALR
DENNIS, F. L.....301 Ferguson Bldg., ALR
KETTELKAMP, F. O.....301 Ferguson Bldg., ALR
NEEPER, EDW. R.....Exchange Nat'l Bank Bldg., Op.

DENVER

ALLEN, J. HENRY.....Republic Bldg., ALR
BANE, W. C.....1005 Republic Bldg., OALR
BANE, WM. M.....1005 Republic Bldg., OALR
BAUM, H. L.....510 Republic Bldg., ALR
BLACK, MELVILLE.....424 Metropolitan Bldg., Op.
BOYD, E. T.....823 Majestic Bldg., OALR
BROWN, MORROW D.....432 Republic Bldg., ALR
CARMODY, T. E.....Metropolitan Bldg., ALR
COLLINS, E. W.....508 Majestic Bldg., ALR
COOPER, C. E.....652 Metropolitan Bldg., ALR
CRISP, WM. H.....Metropolitan Bldg., Op.
DARROW, C. H.....110 Metropolitan Bldg., ALR
FINNOFF, W. C.....Republic Bldg., Op.
FOLEY, J. W.....Majestic Bldg., OALR
GALE, M. JEAN.....Republic Bldg., Op.
GALLAHER, T. J.....California Bldg., ALR

GREENE, LAURENCE W.....Republic Bldg., ALR
HALSTED, FRED S.....738 Metropolitan Bldg., OALR
HICKEY, H. L.....934 Republic Bldg., ALR
JACKSON, EDWARD.....314 14th St., Op.
LAFF, H. I.....Metropolitan Bldg., ALR
LEVY, ROBERT.....Metropolitan Bldg., ALR
LEYDA, JAMES H.....Metropolitan Bldg., ALR
LOCHARD, L. B.....Metropolitan Bldg., ALR
McCAW, JOHN A.....418 Majestic Bldg., Op.
McGILLICUDDY, OLIVER B.....Metropolitan Bldg., ALR
McKEOWN, ELMER E.....Republic Bldg., OALR
MONAGHAN, D. G.....Majestic Bldg., Op.
O'ROURKE, D. H.....217 Imperial Bldg., Op.
SEDWICK, W. A.....Republic Bldg., Op.
SHIELDS, J. M.....264 Metropolitan Bldg., Op.
STRICKLER, D. A.....Republic Bldg., Op.
SWERDFEGER, E. B.....1038 Republic Bldg., ALR
WALKER, C. E.....517 Jacobson Bldg., Op.

GRAND JUNCTION

CARY, G. C.....Canon Block, OALR

GREELEY

ATKINSON, THOMAS ERNEST.....Coronado Bldg., ALR
LEHAN, JAMES W.....Park Place Bldg., OALR
RINGLE, CHAS. A.....Coronado Block, ALR

LEADVILLE

STRONG, J. C.....810 Harrison Ave., OALR

LONGMONT

SIDWELL, CLARENCE EDWARD.....Longmont Hospital, ALR

PUEBLO

HOPKINS, GUY H.....702 N. Main St., Op.
LASSEN, FRITZ.....Pueblo Clinic, OALR

STERLING

PALMER, FRANK E.....301 Main St., OALR

TRINIDAD

RICHIE, L. T.....McCormick Bldg., OALR

CONNECTICUT

BRIDGEPORT

DEWITT, EDWARD NICHOLAS.....836 Myrtle Ave., Op.A
SMITH, DORLAND.....836 Myrtle Ave., Op.A

DANIELSON

BURROUGHS, G. M.....5 Broad St., Op.

HARTFORD

HOGAN, W. L.....750 Main St., OALR
MCCLELLAN, W. E.....50 Farmington Ave., OALR
PRINCE, ALEXANDER L.....36 Pearl St., Op.
SMITH, E. TERRY.....36 Pearl St., Op.
WHALEN, EDWARD J.....750 Main St., ALR

NEW HAVEN

FERGUSON, R. J.....59 College St., OALR
FLYNN, CHAS. T.....41 Thumbull St., ALR
SHEAHAN, WM. L.....59 College St., OALR

NEW LONDON

ROBISON, JAMES T.....309 State St., ALR
WOODRUFF, T. A.....309 State St., Op.

STAMFORD

WHITE, WM. BEVERLY.....813 Gurley Bldg., OALR

WATERBURY

GOOD, WILLIAM MURRAY.....63 Center St., ALR

DELAWARE

WILMINGTON

BONNER, W. F.....224 Del. Trust Bldg., OALR
MAYERBERG, EMIL R.....Medical Arts Bldg., ALR
WHITE, CHAS. P.....507 Citizens Bank Bldg., OALR

DISTRICT OF COLUMBIA

WASHINGTON

ADAMS, ELDRIDGE S.....Aeronautics Branch, Dept. of Commerce, ALR
CONSTANTINOPLE, P. S.....1801 Eye St., N. W., ALR
DABNEY, VIRGINIUS.....1633 Connecticut Ave., ALR
DAVIES, H. F.....1835 Eye St., N. W., ALR
DAVIS, W. T.....927 Farragut Sq., Op.
EARLEY, JAMES H.....1801 Eye St., N. W., ALR
EBERT, EDWIN C.....Naval Dispensary, ALR
FLYNN, JAMES A.....1511 Rhode Island Ave., N. W., OALR
GILL, G. BACHE.....1624 Eye St., N. W., ALR
GREEAR, JAMES NOAH, JR.....1740 M St., N. W., Op.
HENNING, CARL.....1835 Eye St., N. W., Op.
LAMB, ROBT. SCOTT.....Stoneleigh Court, Op.
MUNCASTER, S. B.....1026 16th St., N. W., Op.
NEWELL, WM. S.....1029 Vermont Ave., Op.
PENDEXTER, RALPH S.....1424 K St., N. W., Op.
RUNK, ROXETTE LANDIS.....Wash. Sanit. & Hosp., Takoma Pr., ALR
SCHLANSER, MAJOR A. E.....Walter Reed Gen. Hosp., OALR
SHUTE, D. K.....1727 De Sales St., N. W., Op.
TRIBLE, G. B.....1801 Eye St., N. W., OALR
WILKINSON, OSCAR.....1408 L St., N. W., OALR
ZINKHAN, A. M.....The Rochambeau, ALR

FLORIDA

JACKSONVILLE

BOONE, J. L.....Professional Bldg., ALR
HEGGIE, N. M.....Buckman Bldg., OALR
KNAUER, WM. J.....Buckman Bldg., ALR
PARRAMORE, JAMES BUCHANAN.....St. James Bldg., ALR
RICHARDSON, S. A.....111 W. Adams St., Op.

MIAMI

CHANDLER, G. E.....Huntington Bldg., ALR
DEBOE, M. P.....1st Nat'l Bank Bldg., OALR
HODSON, B. F.....418 Security Bldg., OALR
INGERSOLL, J. M.....1700 S. Bayshore Lane, ALR
WHITAKER, J.....905 Congress Bldg., OALR

MIAMI BEACH

BARNHILL, J. F.....5369 Pine Tree Drive, ALR
PALMER, BASCOM H.....502 Huntington Bldg., OALR
REPASS, R. E.....337 Lincoln Road, ALR

ORLANDO

INGRAM, L. C.....Orlando Clinic, OALR
SHOEMAKER, SAMUEL A.....30 E. Church St., OALR

PENSACOLA

LISCHKOFF, M. A.....Blount Bldg., OALR

ST. PETERSBURG

HEARD, MARY K.....128 19th Ave. North, Op.

TAMPA

FARRIOR, J. BROWN.....605 Citizens' Bank Bldg., OALR
FORBES, SHERMAN B.....409 Citizens' Bank Bldg., OALR
LOWRY, B. W.....205 Zack St., OALR

WEST PALM BEACH

MILLER, ALICE R.....418 Fern St., OALR

WINTER PARK

OESTERLING, H. E.....642 N. Interlachen Ave., OALR

GEORGIA

ATLANTA

BRAWNER, LEON EDWARD.....157 Forrest Ave., N. E., ALR
CALHOUN, F. PHINIZY.....486 Peach Tree St., N. E., Op.
CLAY, G. E.....Medical Arts Bldg., Op.
FORT, A. G.....303 Medical Arts Bldg., OALR
LOKEY, H. M.....Medical Arts Bldg., OALR
MARTIN, WM. O. JR.....Medical Arts Bldg., Op.
WARREN, WM. C. JR.....Atlanta Nat'l Bank Bldg., ALR

AUGUSTA

OERTEL, T. E.....S. F. Bldg., OALR

ROME

SMITH, GEORGE B.....Harbin Hospital, OALR

VALDOSTA

SMITH, J. M.....134½ N. Patterson St., OALR

HAWAII

HONOLULU

MORGAN, JAMES A.....48 Young Bldg., OALR
PINKERTON, FORREST JOY.....46 Young Hotel Bldg., OALR
VAN POOLE, G. McD.....Young Bldg., OALR

IDAHO

NAMPA

SHAWHAN, GLENN E.....Smallwood Bldg., OALR

POCATELLO

CLOTHIER, JOSEPH.....Kane Bldg., OALR

TWIN FALLS

WETHERBEE, CHARLES.....Booth Bldg., OALR

ILLINOIS

ALTON

JONES, F. W.....Times Bldg., OALR
MIDDLETON, HARRY E.....103 East Broadway, OALR

AURORA

DARMER, GEORGE A.....401 Terminal Bldg., OALR

BELLEVILLE

BIRSNER, LOUIS J.....25 1st Nat'l Bank Bldg., ALR

BLOOMINGTON

FOX, RALPH D.....Unity Bldg., OALR
GAILEY, WATSON W.....Griesheim Bldg., OALR
WATKINS, HAROLD R.....Griesheim Bldg., ALR

CHAMPAIGN

FORD, HANBY LEWIS.....401 Robeson Bldg., OALR

CHICAGO

ALLEN, THOMAS DYER.....122 S. Michigan Blvd., Op.
ANDREWS, A. H.....30 N. Michigan Blvd., OALR
ANDREWS, B. F.....25 E. Washington St., OALR

BALLENGER, H. C. ..... 25 E. Washington St., ALR
BECK, JOSEPH C. ..... 185 N. Wabash Ave., ALR
BEEM, IONE F. ..... 31 N. State St., OALR
BERNHEIMER, B. L. ..... 104 S. Michigan Ave., ALR
BOTHMAN, LOUIS ..... 122 S. Michigan Ave., Op.
BRAWLEY, F. E. ..... 30 N. Michigan Blvd., Op.
BREDLAU, ERNEST A. ..... 2753 North Ave., ALR
CARNCROSS, HELEN ..... 58 E. Washington St., Op.
CAVANAUGH, J. A. ..... Suite 1712-30 N. Michigan Blvd., ALR
CHRISTOPH, CARL H. ..... 30 N. Michigan Blvd., OALR
CLEFF, OSCAR ..... 55 E. Washington St., OALR
CLEMENT, C. C. ..... 30 N. Michigan Blvd., OALR
CROSSLEY, E. R. ..... 30 N. Michigan Blvd., OALR
CUSHMAN, BEULAH ..... 25 E. Washington St., Op.
DAVIDSON, H. P. ..... 58 E. Washington St., Op.
DELPH, JOHN F. ..... 104 S. Michigan Blvd., ALR
DENNIS, GEORGE J. ..... 25 E. Washington St., ALR
FAITH, THOMAS ..... 31 N. State St., OALR
FARRELL, P. J. H. ..... 25 E. Washington St., OALR
FINDLAY, E. K. ..... 30 N. Michigan Ave., Op.
FISHER, W. A. ..... 231 W. Washington St., OALR
FISKE, GEORGE F. ..... 25 E. Washington St., OALR
FITZGERALD, J. P. ..... 30 N. Michigan Blvd., Op.
FOLK, MAX LYON ..... 55 E. Washington St., OALR
FOWLER, EARL B. ..... 55 E. Washington St., Op.
FOX, NOAH ..... 841 E. 63rd St., ALR
FRANK, IRA ..... 104 S. Michigan Blvd., ALR
FULLER, HARRY B. ..... 2219 E. 70th Place, OALR
GAMBLE, RICHARD COTTER ..... 30 N. Michigan Ave., Op.
GARRAGHAN, EDW. F. ..... 25 E. Washington St., OALR
GEIGER, ARTHUR HENRY ..... 554 Fullerton Parkway, ALR
GIFFORD, SANFORD R. ..... 58 E. Washington St., Op.
GRADLE, H. S. ..... 58 E. Washington St., Op.
GRIM, U. J. ..... 30 N. Michigan Blvd., OALR
GUTTMAN, MELVIN REESE ..... 185 N. Wabash Ave., ALR
HALL, ALICE K. ..... 25 E. Washington St., ALR
HASELTINE, BURTON ..... 122 S. Michigan Ave., OALR
HAWLEY, C. W. ..... Medical Arts Bldg., OALR
HAYDEN, AUSTIN A. ..... Suite 2009, 25 E. Washington St., OALR
HOFFMAN, L. G. ..... 30 N. Michigan Blvd., OALR
JORDAN, GEORGE T. ..... 30 N. Michigan Blvd., OALR
KAHN, MYRON E. ..... 104 S. Michigan Ave., ALR
KILLEEN, JOHN J. ..... 104 S. Michigan Blvd., ALR
KRAFT, OSCAR H. ..... 1518 N. Dearborn Parkway, Op.
LEBENSOHN, JAMES E. ..... 25 E. Washington St., OALR
LEDERER, FRANCIS L. ..... 30 N. Michigan Blvd., ALR
LEECH, VERNON M. ..... 55 E. Washington St., Op.
LIFSCHUTZ, JACOB ..... 310 S. Michigan Blvd., OALR
LIVINGSTON, GEORGE S. ..... 185 N. Wabash Ave., ALR

LORING, J. B.....25 E. Washington St., OALR
LYONS, H. R.....30 N. Michigan Blvd., ALR
MCBEAN, GEORGE M.....58 E. Washington St., OALR
MCGINNIS, EDWIN.....104 S. Michigan Blvd., ALR
MARQUIS, GEORGE P.....30 N. Michigan Blvd., ALR
MAYER, LEO LOEB.....104 S. Michigan Ave., Op.
MEYER, SAMUEL J.....58 E. Washington St., Op.
MUNDT, G. H.....25 E. Washington St., OALR
MURRAY, A. N.....4753 Broadway, OALR
NANCE, WILLIS O.....30 N. Michigan Blvd., OALR
NOONAN, W. J.....4753 Broadway, OALR
NORCROSS, E. P.....8 S. Michigan Blvd., ALR
NOVAK, FRANK J. JR.....1712, 30 N. Michigan Blvd., ALR
NUGENT, OSCAR B.....231 W. Washington St., Op.
ORCUTT, D. C.....30 N. Michigan Blvd., Op.
PEARLMAN, S. J.....180 N. Michigan Ave., ALR
POLLOCK, H. L.....2551 N. Clark St., ALR
SALINGER, SAMUEL.....25 E. Washington St., ALR
SHAPIRO, S. L.....25 E. Washington St., ALR
SONNENSCHEIN, R.....180 N. Michigan Blvd., ALR
SPIESMAN, IRWIN GABRIEL.....25 E. Washington St., ALR
STRAUSS, J. F.....104 S. Michigan Blvd., ALR
SUKER, GEORGE F.....25 E. Washington St., Op.
TAYLOR, LILLIAN E.....25 E. Washington St., ALR
THEOBALD, JOHN JOSEPH.....58 E. Washington St., ALR
THEOBALD, W. H.....307 N. Michigan Ave., ALR
TIVNEN, RICHARD J.....30 N. Michigan Blvd., OALR
VAN DELLEN, A. L.....6857 S. Halsted St., Op.
VON COLDITZ, G. THOMSEN.....25 E. Washington St., OALR
VON DER HEYDT, ROBERT.....25 E. Washington St., Op.
WAGNER, CARL B.....30 N. State St., OALR
WALKER, HERBERT S.....30 N. Michigan Ave., OALR
WILDER, WM. H.....122 S. Michigan Blvd., Op.
WOLF, SAMUEL.....55 E. Washington St., Op.
WOOD, CASEY A.....7 W. Madison St., Op.

CHICAGO HEIGHTS

GORRELL, T. J. H.....16th and Vincennes Ave., OALR

DANVILLE

BALDWIN, H. E.....Temple Bldg., ALR
CARMODY, THOMAS J.....408 The Temple Bldg., OALR

DIXON

MCNICHOLS, W. A.....Dixon Nat'l Bank Bldg., OALR

EAST ST. LOUIS

HANSON, WM. LUDWIG.....318 Murphy Bldg., ALR
SPITZE, E. C.....Murphy Bldg., Op.

EVANSTON

BOOT, G. W.....811 Sherman Ave., OALR
DODD, OSCAR.....636 Church St., Op.
GALLOWAY, T. C.....636 Church St., ALR
SWAN, C. J.....636 Church St., OALR

FREEPORT

CLARK, J. SHELDON.....509 State Bank Bldg., OALR

GALESBURG

MATHENY, R. C.....306 E. Main St., OALR

GREAT LAKES

WEBER, H. C.....U. S. Naval Hospital, OALR

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EAREL, A. M.....Willdon Bldg., OALR

HUBBARD WOODS

LOEB, CLARENCE.....1054 Tower Road, Op.

JACKSONVILLE

ADAMS, A. L.....Professional Bldg., OALR

JOLIET

WOODRUFF, H. W.....500 N. Ottawa St., OALR

KANKAKEE

GEIGER, C. W.....Arcade Bldg., OALR

LA SALLE

WOODS, R. H.....Neustadt Bldg., Op.

MACOMB

DUNTLEY, G. S.....103 S. Side Sq., OALR

MATTOON

VOIGT, C. B.....1702 Broadway, OALR

MOLINE

FLATLEY, T. J.....1314 Fifth Ave., OALR

OAK PARK

THEOBALD, GEORGIANA DVORAK.....715 Lake St., Op.

PEKIN

GLASFORD, S. T.....Farmers' Bank Bldg., OALR

PEORIA

BLICKENSTAFF, A. J.....Peoria Life Bldg., OALR
BROBST, C. H.....Central Bank Bldg., OALR
DUANE, J. F.....Jefferson Bldg., OALR

FLOYD, T. W.....811 Peoria Life Bldg., OALR
THOMAS, C. D.....Central National Bank Bldg., OALR
WILLIAMS, W. C.....Jefferson Bldg., OALR

PONTIAC

MIDDLETON, A. B.....Sterry Blk., OALR

QUINCY

GABRIEL, C. K.....W. C. U. Bldg., ALR
MERCER, RAY.....W. C. U. Bldg., OALR
STEVENSON, WALTER.....W. C. U. Bldg., OALR

ROCKFORD

FRINGER, W. R.....William Brown Bldg., Op.
WORMLEY, H. R.....Talcott Bldg., OALR

SPRINGFIELD

DEAL, JOHN FRANCIS.....First Nat'l Bank Bldg., OALR
HAGLER, E. E.....401 East Capitol Ave., OALR

STERLING

BRODERICK, FRANK W.....Central Trust Bldg., OALR

STREATOR

LESTER, HARRY S.....420 Murray Bldg., OALR

INDIANA

CRAWFORDSVILLE

LINGEMAN, B. N.....Ben Hur Bldg., ALR

ELKHART

PORTER, JESSE B.....415 S. Second St., OALR

EVANSVILLE

BROSE, L. D.....501 S. East First St., OALR
DYER, WALLACE CURTIS.....Central Union Bank Bldg., ALR
KNAPP, B. J.....Citizens' Bank Bldg., OALR
RAVDIN, B. D.....Central Union Bank Bldg., OALR
RAVDIN, M.....Central Union Bank Bldg., OALR
STANTON, HARMON LAM.....Cleveland Life Bldg., OALR

FORT WAYNE

BULSON, A. E.....406 W. Berry St., OALR
BULSON, EUGENE LORING.....406 W. Berry St., OALR
MCBRIDE, W. O.....2902 S. Fairchild, Ave., OALR

HAMMOND

KUHN, HEDWIG S.....First Trust Bldg., Op.
KUHN, HUGH A.....First Trust Bldg., ALR
LLOYD, A. W.....804 Lloyd Bldg., OALR
SHANKLIN, E. M.....575 Hohman St., OALR

INDIANAPOLIS

ADAMS, D. S.....508 Hume-Mansur Bldg., OALR
ARONSON, SIDNEY S.....23 E. Ohio St., ALR
BERRY, D. F.....Hume-Mansur Bldg., ALR
CARMACK, J. W.....Hume-Mansur Bldg., ALR
CHAPPELL, R. S.....305 Traction Terminal Bldg., ALR
CLEVENGER, W. F.....Hume-Mansur Bldg., ALR
CRAFT, KENNETH L.....Hume-Mansur Bldg., ALR
DEWEY, C. H., MAJ., M. C.,
U. S. Army Hospital, Ft. Benjamin Harrison, OALR
HUGHES, W. F.....401 Hume-Mansur Bldg., Op.
KEARBY, D. O.....709 Hume-Mansur Bldg., ALR
LARKIN, BERNARD J.....Hume-Mansur Bldg., Op.
LINGEMAN, EDWARD LAUREL.....406 Pennway Bldg., ALR
MCCASKEY, C. H.....Continental Bank Bldg., ALR
MASTERS, ROBERT J.....Hume-Mansur Bldg., Op.
MOLT, WM. F.....529 Bankers Trust Bldg., ALR
NEWCOMB, JOHN R.....410 Hume-Mansur Bldg., Op.
OVERMAN, F. V.....Hume-Mansur Bldg., ALR
PFAFFLIN, C. A.....445 Banker Trust Bldg., OALR
ROW, GEORGE S.....906 Hume-Mansur Bldg., Op.
TOMLIN, W. S.....Hume-Mansur Bldg., ALR
WRIGHT, J. W. M.....Hume-Mansur Bldg., ALR

KOKOMO

ADAMS, CHAS. JOSEPH.....101 W. Sycamore St., OALR

LOGANSPORT

EGAN, BURTON WRIGHT.....408 North St., OALR

MARION

BRAUNLIN, WM. H.....Marion Nat'l Bank Bldg., OALR
MILLER, C. E.....Western Reserve Bldg., OALR

MICHIGAN CITY

KRIEGER, G. M.....701 Washington St., OALR

MUNCIE

MILLER, C. E.....917 E. Main St., OALR

SOUTH BEND

BOONE, J. C.....213 Pythian Bldg., ALR
BOYD-SNEE, HARRY.....716 J. M. S. Bldg., ALR
CASSADY, J. VERNAL.....654 Associate Bldg., OALR
EDWARDS, SLOCOMB R.....Christman Bldg., OALR
LENT, E. J.....122 N. Lafayette Blvd., OALR
SAVERY, C. E.....206 Sherland Bldg., OALR

TERRE HAUTE

ALLEN, ORRIS T.....422 Rose Dispensary, OALR
GILLUM, J. R.....221 S. 6th St., OALR
STEWART, WALTER E.....Tribune Bldg., OALR

WARSAW

HOWARD, C. N.....Warsaw Clinic, OALR

IOWA

AMES

DYER, BENJAMIN G.....ALR

BURLINGTON

LA FORCE, E. F.....Iowa State Bank Bldg., OALR
YOUNG, H. B.....204 Jefferson St., OALR

CEDAR RAPIDS

BAILEY, FRED W.....Merchants Nat'l Bank Bldg., OALR
IVINS, H. M.....815 Merchants Nat'l Bank Bldg., OALR

CLINTON

WEIH, E. P.....Wilson Bldg., OALR

COUNCIL BLUFFS

DEAN, F. W.....532 First Ave., OALR
HOWARD, LLOYD G.....Council Bluffs Clinic, OALR
MAIDEN, S. D.....403 Bennett Bldg., OALR

CRESTON

CHILDS, H. A.....Eagle Blk., OALR

DAVENPORT

HANDS, S. GILLOTT.....201 West Second St., ALR
HARKNESS, GORDON F.....Putnam Bldg., OALR
HOFMANN, W. P.....707 Kahl Bldg., OALR
LAMB, HARRY HOWE.....American Bank Bldg., ALR

DES MOINES

JONES, CECIL C.....1120 Equitable Bldg., OALR
PEARSON, WM. W.....417 Bankers Trust Bldg., OALR

DUBUQUE

THOMPSON, HOWARD E.....1100 Main St., OALR

FORT DODGE

CHASE, S. B.....Carver Bldg., OALR

INDEPENDENCE

AGNEW, F. F.....120 Third Ave., S. E., OALR

IOWA CITY

BOILER, W. F.....Dey Bldg., OALR
LIERLE, DEAN MCA.....University Hospital, ALR
MOORE, PAUL MCNARY, JR.....University Hospital, ALR
O'BRIEN, C. S.....University Hospital, Op.
RUTHERFORD, C. W.....Univ. Hosp., Dept. Ophth., Op.

KEOKUK

LAPSLEY, ROBERT M.....Masonic Temple, OALR

LE MARS

JOYNT, M. J.....Royal Bldg., ALR

MARSHALLTOWN

COBB, EDWIN.....Masonic Temple Bldg., OALR
FRENCH, R. F.....Masonic Temple Bldg., OALR
SINGLETON, E. M.....111 E. Church St., R
WOLFE, OTIS R.....415 Masonic Temple, OALR

MASON CITY

FALLOWS, HOWARD D.....Park Hospital Clinic, OALR
O'BRIEN, STEVE A.....First Nat'l Bank Bldg., OALR

MUSCATINE

CONE, ALFRED JARED.....404 Laurel Bldg., ALR

NEWTON

ENGLE, H. P.....Maytag Bldg., OALR
GREENWOOD, G. J.....317 First Ave., East, ALR

OTTUMWA

TAYLOR, CHARLES B.....118 E. Second St., ALR

SHENANDOAH

WEAVER, JOHN O.....608½ W. Sheridan Ave., ALR

SIOUX CITY

DECKER, J. C.....Frances Bldg., OALR
FRANCHERE, FRED'K. E.....Toy Bank Bldg., OALR
GITTINS, T. R.....401 Davidson Bldg., OALR
NAFTZGER, J. B.....Davidson Bldg., OALR
REEDER, J. E.....Davidson Bldg., OALR
ROOST, F. H.....OALR
SALLANDER, F. W.....New Orpheum Bldg., OALR
SECOY, F. L.....301 Davidson Bldg., OALR
TRIPP, LEROY R.....Security Bldg., OALR

WATERLOO

BENDER, H. A.....Black Bldg., ALR
JOYNT, A. J.....Black Bldg., OALR
REULING, F. H.....Black Bldg., OALR
RUSSELL, R. E.....601 Pioneer Bank Bldg., OALR

WAVERLY

ROBINSON, R. E.....205½ E. Bremer Ave., OALR

KANSAS

ATCHISON

BRIBACH, EUGENE J.....212 Simpson Bldg., OALR

COFFEYVILLE

CHADWICK, IRA B.....715 Walnut St., Op.

CONCORDIA

ROBERTSON, EDW. N.....132½ W. 6th St., OALR

ELDORADO

SHULTS, E. J. G.....Haines Bldg., OALR

EMPORIA

GRANGER, W. B.....Citizens Nat'l Bank Bldg., OALR
TRIMBLE, CLARENCE S. .....405 Citizens Nat'l Bank Bldg., OALR

HUTCHINSON

GREEVER, B. L.....610 Rorabaugh-Wiley Bldg., OALR
SCALES, H. L.....Rorabaugh-Wiley Bldg., OALR
SCHRANT, J. H.....14 West First St., OALR

KANSAS CITY

LIDIKAY, C. J.....903 N. Seventh St., OALR
SPAKE, LAVERNE B.....322 Brotherhood Bldg., ALR

LAWRENCE

POWELL, LYLE S.....813 Mass. St., OALR

LEAVENWORTH

HAAS, FREDERICK J.....Masonic Bldg., OALR

PRATT

JENKINS, MIKE CLEMONT.....223 S. Main St., ALR

TOPEKA

ALKIRE, H. L.....614 Kansas Ave., OALR
ALLEN, G. HENRY.....Mills Bldg., OALR
ALLEN, GEORGE V.....835 Kansas Ave., OALR
REED, WM. W.....Mills Bldg., ALR

WELLINGTON

DILLON, IRA HUGH .....Hatcher Hospital, Op.

WICHITA

BROWNELL, MORTON EMMONS.....1019 First Nat'l Bank Bldg., Op.
CARTER, E. D.....Central Bldg., ALR
CHENEY, JAMES W.....702 Schweiter Bldg., OALR
GILLETT, WILBUR G.....201½ N. Main St., Op.
GSELL, J. F.....911 Beacon Bldg., OALR

MAGGARD, D. I. ..... 715 Orpheum Bldg., OALR
PALMER, EDW. M. ..... 629 Beacon Bldg., OALR
SEYDELL, E. M. ..... 1030 First Nat'l Bank Bldg., ALR
WEAVER, T. W. ..... 201½ N. Main St., Op.

WINFIELD

RALLS, C. T. ..... First National Bank Bldg., OALR

KENTUCKY

BEREA

COWLEY, ROBERT HENRY ..... OALR

COVINGTON

BLEDSOE, R. W. ..... 1005 Madison Ave., OALR

FULTON

COHN, SELDON ..... OALR

HARLAN

GUNN, MILUS LYDELL ..... Smith Bldg., ALR

LEXINGTON

ARMSTRONG, R. M. ..... 1010 Fayette Bank Bldg., OALR
DEWEESE, CLARENCE ..... 201 West Second St., OALR
MARKS, SAMUEL BLACKBURN ..... Security Trust Bldg., ALR
OFFUTT, W. N. ..... 406 Security Trust Bldg., OALR
STUCKY, J. A. ..... Fayette Nat'l Bank Bldg., OALR

LOUISVILLE

BASS, A. L. ..... Heyburn Bldg., OALR
DABNEY, S. G. ..... Starks Bldg., OALR
DEAN, WALTER ..... 300 Francis Bldg., OALR
DULANEY, OCTAVIUS ..... 811 Brown Bldg., ALR
HALL, G. C. ..... 705 Brown Bldg., OALR
PFINGST, A. O. ..... Heyburn Bldg., Op.
SHAFER, J. J. ..... Starks Bldg., OALR
VAUGHAN, G. E. ..... 711 Third St., OALR
WOLFE, CLAUDE T. ..... Heyburn Bldg., OALR

NEWPORT

THOMASSON, W. J. ..... 942 York St., ALR

PADUCAH

PACE, ERRETT ..... City National Bank Bldg., ALR
REYNOLDS, H. G. ..... City National Bank Bldg., OALR

RICHMOND

DUNN, MURISON ..... OALR

LOUISIANA

NEW ORLEANS

BAHN, CHAS. A.....1707 Pere Marquette Bldg., Op.
BLUM, HENRY N.....1211 Maison Blanche Bldg., Op.
COLLIER, GEO. BENJAMIN.....1317 Whitney Bldg., ALR
FUCHS, VALENTINE HENRY.....Chaille Bldg., ALR
HASPEL, M. DAVID.....603 Maison Blanche Bldg., OALR
LANDFRIED, C. J.....607 Baronne Bldg., ALR
LEJEUNE, F. E.....632 Maison Blanche Bldg., ALR
LYNCH, R. C.....Maison Blanche Bldg., ALR
SMITH, VICTOR C.....1707 Pere Marquette Bldg., Op.
TAQUINO, GEORGE J.....208 Audubon Bldg., ALR
WAGNER, W. A.....American Bank Bldg., ALR
WEIL, A. I.....3503 Prytania St., ALR

SHREVEPORT

LARUE, C. L.....Highland Clinic, OALR
MOISE, ALLYN B.....Medical Arts Bldg., ALR
SCALES, J. L.....Medical Arts Bldg., OALR

MAINE

AUGUSTA

TURNER, OLIVER W.....Masonic Temple, OALR

PORTLAND

BEACH, S. J.....704 Congress St., Op.
CUMMINGS, GEORGE O.....47 Deering St., ALR
GILBERT, FRANK Y.....148 Park St., OALR
GORDON, CHAS. H.....46 Deering St., ALR
HASKELL, ALFRED W.....129 Park St., Op.
HOLT, E. E., JR.....723 Congress St., Op.
O'SULLIVAN, T. J.....10 Pine St., ALR
SPALDING, J. A.....627 Congress St., Op.A

WATERVILLE

HILL, FREDERICK T.....Professional Bldg., ALR
HILL, HOWARD F.....Professional Bldg., Op.

MARYLAND

ANNAPOLIS

ALBRIGHT, LT.-COM. P. M., M. C., U. S. N.
U. S. Naval Academy, OALR
WELCH, ROBERT S. G.....46 State Circle, OALR

BALTIMORE

BORDLEY, JAMES, JR.....330 N. Charles St., OALR
FRIEDENWALD, HARRY.....1212 Eutaw Pl., Op.A
FRIEDENWALD, J. S.....1212 Eutaw Pl., Op.
GOLDBACK, L. J.....1012 N. Charles St., Op.

KEMLER, JOS. ..... 1908 Eutaw Pl., OALR
LOOPER, EDWARD ANDERSON ..... 104 Madison St., OALR
PETERMAN, H. E. ..... 518 Cathedral St., OALR
SAVAGE, M. M. ..... 1720 Eutaw Pl., OALR
TARUN, WILLIAM ..... 104 W. Madison St., OALR
WOODS, HIRAM ..... 842 Park Ave., Op.A

CUMBERLAND

JONES, E. L. ..... First National Bank Bldg., OALR
ROBINSON, H. T. ..... Medical Bldg., OALR
SHARRETT, GEORGE O. ..... 119 Bedford, OALR

MASSACHUSETTS

BEVERLY

ODENEAL, THOMAS HELM ..... 234 Cabot St., OALR

BOSTON

BRYANT, ALICE GERTRUDE ..... 502 Beacon St., ALR
CARVILL, MAUD ..... 82 Commonwealth Ave., Op.
CHENERY, W. E. ..... 377 Commonwealth Ave., ALR
CORBETT, J. J. ..... 520 Beacon St., OALR
DERBY, GEORGE S. ..... 5 Bay State Road, Op.
FREEDMAN, L. M. ..... 475 Commonwealth Ave., ALR
GREENWOOD, ALLEN ..... 82 Commonwealth Ave., Op.
HURLEY, E. D. ..... 419 Boylston St., Op.
INGLIS, HARRY J. ..... 43 Bay State Road, ALR
KLEINERT, MARGARET NOYES ..... 82 Commonwealth Ave., ALR
KNOWLTON, CHARLES DAVISON ..... 520 Commonwealth Ave., ALR
LANCASTER, W. B. ..... 520 Commonwealth Ave., Op.
LEWIS, J. P. ..... 543 Boylston St., ALR
LURIE, MOSES H. ..... 483 Beacon St., ALR
MOSHER, HARRIS P. ..... 828 Beacon St., ALR
PORTER, CHAS. T. ..... 520 Commonwealth Ave., ALR
QUINCY, J. E. ..... 270 Commonwealth Ave., ALR
RICE, G. B. ..... 99 Bay State Road, ALR
RICHARDS, L. G. ..... 319 Longwood Ave., ALR
ROWLAND, W. D. ..... 220 Clarendon St., Op.
SCHALL, L. A. ..... 270 Commonwealth Ave., ALR
SKIRBALL, J. J. ..... 353 Commonwealth Ave., OALR
THOMPSON, PETER H. ..... 308 Commonwealth Ave., Op.
TINGLEY, LOUISA PAINE ..... 9 Massachusetts Ave., Op.
TOBEY, GEORGE L., JR. ..... 270 Commonwealth Ave., ALR
WALKER, WALDO W. ..... 99 Bay State Road, ALR
WELLS, DAVID W. ..... Hotel Westminster, Op.

CLINTON

MONAHAN, J. A. ..... 181 Chestnut St., OALR

FALL RIVER

RYDER, D. R. ..... 151 Rock St., ALR

GARDNER

BONE, HERMAN D.....19 Pleasant St., OALR

HOLYOKE

BURKE, JOHN R.....207 Elm St., OALR
HUSSEY, E. J.....245 Maple St., OALR

LAWRENCE

CONLON, F. A.....301 Essex St., OALR
MERRILL, WM. HOWE.....Bay State Bldg., OALR

LOWELL

WIGGIN, WM. IRVING.....202 Central St., ALR

LYNN

JONES, E. W.....44 Atlanta St., Op.A
O'REILLY, W. F.....44 S. Common St., OALR

NEW BEDFORD

POTTER, L. F. .....68 Walnut St., OALR

NORTHAMPTON

COLLINS, J. D.....187 Main St., ALR
DOW, FRANK E.....4 West St., Op.

PALMER

MOORE, G. A.....353 Main St., OALR

PITTSFIELD

THOMPSON, G. H.....74 North St., OALR

SALEM

HENNESSEY, W. W.....333 Essex St., OALR

SPRINGFIELD

BYRNES, H. F.....67 Chestnut St., OALR
CARLETON, RALPH.....10 Temple St., Op.
GALLAGHER, J. H. C. .....1537 Main St., ALR
IRWIN, VINCENT J., JR.....1634 Main St., OALR
IRWIN, V. J., SR. .....1634 Main St., OALR

TAUNTON

BAKER, HARRY B.....59 Main St., OALR

WESTFIELD

DECHTER, M. ARCHIBALD .....Professional Bldg., ALR

WORCESTER

BERRY, GORDON.....36 Pleasant St., ALR
CAHILL, J. W.....390 Main St., OALR
CROSS, A. E.....390 Main St., Op.

ESTABROOK, CHAS. T.....857 Pleasant St., OALR
MESSIER, A. E.....390 Main St., OALR
RICE, JOHN E.....390 Main St., OALR

MICHIGAN

ANN ARBOR

FURSTENBERG, A. C.....First Nat'l Bank Bldg., ALR
SLOCUM, GEORGE.....First National Bank Bldg., Op.

BATTLE CREEK

COLVER, B. N.....Sanitarium 1, ALR
FRASER, R. H.....Battle Creek Sanitarium, ALR
HAUGHEY, WILFRED H.....303 Post Bldg., OALR
SLEIGHT, R. D.....303 Post Bldg., Op.
WENCKE, CARL G.....Battle Creek Sanitarium, ALR

BAY CITY

BAKER, CHAS. H.....Bay City Bank Bldg., OALR

DETROIT

AMBERG, EMIL.....622 Maccabee Bldg., ALR
BARNETT, SAUL E.....25 Parsons St., ALR
BEATTIE, ROBERT.....David Whitney Bldg., OALR
BEGLE, HOWELL L.....2730 East Jefferson Ave., Op.
BENTLEY, NEIL I.....1161 David Whitney Bldg., OALR
BERNSTEIN, E. J.....10 Peterboro, OALR
BROWN, AUDREY O.....742 Maccabee Bldg., OALR
CAMPBELL, D. A.....David Whitney Bldg., OALR
CAMPBELL, DON M.....David Whitney Bldg., OALR
CAMPBELL, M. D.....David Whitney Bldg., OALR
CARTER, JOHN M.....David Whitney Bldg., ALR
CLIFFORD, T. P.....David Whitney Bldg., OALR
COHOE, D. A.....13535 Woodward Ave., ALR
CONNOR, RAY.....1410 Stroh Bldg., OALR
DEFNET, WM. A.....28 Adams Ave. West, ALR
FOWLER, WM.....1048 Maccabee Bldg., OALR
GLOWACKI, BEN FRANCIS.....Maccabee Bldg., OALR
GOUX, RAYMOND SALOT.....545 David Whitney Bldg., OALR
GRANT, LEE E.....David Whitney Bldg., OALR
HALE, ARTHUR STANLEY.....1609 Eaton Tower, OALR
HARRELL, VOSS.....Hofman Bldg., ALR
HEATH, PARKER.....David Whitney Bldg., Op.
HUGHES, RAY W.....1501 David Whitney Bldg., OALR
KEATING, T. F.....1042 Maccabee Bldg., OALR
KREUTZ, GEORGE C.....Henry Ford Hospital, ALR
MCCLELLAND, CARL C.....1857 David Whitney Bldg., OALR
MAIRE, LEWIS E.....710 Kresge Bldg., OALR
MERCER, R. E.....David Whitney Bldg., LR
MERKEL, CHARLES CARSON.....David Whitney Bldg., ALR

MINER, S. G.....2641 E. Jefferson Ave., ALR
MORTON, J. B.....90 E. Warren Ave., OALR
MUNSON, F. T.....2201 E. Jefferson Ave., ALR
PARKER, WALTER R.....David Whitney Bldg., Op.
PEIRCE, HORACE W.....704 Medical Arts Bldg., ALR
POOS, E. E.....Fisher Bldg., OALR
POTTER, WILLIS A.....1355 David Whitney Bldg., ALR
RENAUD, G. L.....510 Joseph Campan Ave., OALR
RYERSON, F. L.....Fine Arts Bldg., OALR
SANDERSON, H. H.....David Whitney Bldg., OALR
SHURLY, BURT R.....62 Adams Ave. W., ALR
SIMPSON, H. LEE.....1409 David Whitney Bldg., ALR
SISSON, RAYMOND J.....753 David Whitney Bldg., Op.
SUMMERS, WM. S.....1613 David Whitney Bldg., OALR
SUTHERLAND, JACOB MARION.....General Motors Bldg., ALR
WALKER, CHAS. C.....604 Maccabee Bldg., Op.
WENDEL, J. S.....744 David Whitney Bldg., ALR
WHITNEY, E. L.....Henry Ford Hospital, ALR
WILLSON, W. W.....2204 Eaton Tower, OALR
WOODWORTH, WM. P.....1424 Maccabee Bldg., OALR
WILKINSON, A. P.....664 Fisher Bldg., ALR

ESCANABA

BOYCE, W. B.....1107 Ludington St., OALR

GRAND RAPIDS

DEAN, ALFRED.....308 Medical Arts Bldg., Op.
GRANT, P. T.....1407 Grand Rapids Nat'l Bank Bldg., OALR
HEETDERKS, D. R.....Medical Arts Bldg., ALR
MCRAE, J. H.....Grand Rapids Clinic, Op.
ROBERTSON, F. D.....Ashton Bldg., OALR
ROLLER, LOUIS A.....446 Paris Ave., S. E., Retired
SNAPP, CARL F.....Medical Arts Bldg., ALR

IONIA

SEDGWICK, O. W.....OALR

IRON MOUNTAIN

BOYCE, G. H.....First Nat'l Bank Bldg., OALR

JACKSON

HARDIE, GEORGE C.....290 W. Michigan Ave., ALR
MCGARVEY, WM. EDWARD.....Jackson City Bank, ALR
WINTER, GEORGE E.....Sun Bldg., OALR

KALAMAZOO

FULKERSON, C. B.....308 Kalamazoo National Bank Bldg., OALR
GRANT, FRED E.....Kalamazoo National Bank Bldg., OALR
WILBUR, EDWARD P.....Bank of Kalamazoo Bldg., OALR

LANSING

BURDICK, AUSTIN F.....416 Tussing Bldg., OALR
ELLIS, CHARLES WARD.....215 N. Walnut St., ALR
WETZEL, JOHN O.....404 Capitol Saving and Loan Bldg., Op.

MENOMINEE

ELWOOD, CALVIN R.....Spies Bldg., OALR
JONES, WM. S.....522 Sheridan Road, OALR

MT. CLEMENS

CROMAN, JOS. M., JR.....115 Cass Ave., OALR

PORT HURON

FRASER, ROBERT C.....940 Military St., ALR

ST. JOSEPH

MCDERMOTT, JOHN J.....1821 Niles Ave., OALR

SAGINAW

CORTOPASSI, ANDRE J.....704 Second Nat'l Bank Bldg., ALR
MCKINNEY, A. R.....704 Second Nat'l Bank Bldg., OALR
ROGERS, A. S.....105 S. Jefferson Ave., OALR
WATSON, R. S.....120 N. Michigan Ave., OALR

TRAVERSE CITY

HOLDSWORTH, FRANK.....Peoples Savings Bank Bldg., OALR

MINNESOTA

ALBERT LEA

FOLKEN, FRANK GARRETT.....306 Albert Lea State Bank Bldg., OALR

BRECKENRIDGE

RICE, CLARENCE PRENTICE.....OALR

CROOKSTON

MORLEY, GEORGE A.....Crookston State Bank Bldg., Op.
OPPEGAARD, C. L.....220 S. Broadway, ALR
PARSONS, J. G.....Crookston Clinic, OALR

DULUTH

KNAPP, F. N.....Bradley Bldg., OALR
SHASTID, T. H.....Sellwood Bldg., Op.
TILDERQUIST, D. L.....425 Bradley Bldg., OALR
WINTER, JOHN A.....205 W. Second St., OALR

FERGUS FALLS

KITTELSON, THEODORE.....Iverson and Lee Bldg., OALR

HIBBING

MORSMAN, L. W.....Morsman Clinic, Op.
RUBY, F. M.....1940 E. Third Ave., OALR
SINAMARK, ANDREW.....205 Power Bldg., OALR

MINNEAPOLIS

BORGESON, E. J. ..... Medical Arts Bldg., Op.
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CAMP, WALTER E. ..... Medical Arts Bldg., OALR
CAMPBELL, ROBERT A. ..... 520 La Salle Bldg., OALR
CLARK, H. S. ..... Medical Arts Bldg., OALR
DORGE, RICHARD IRVING ..... 2319 6th St., OALR
FINK, WALTER H. ..... Medical Arts Bldg., OALR
HANSEN, ERLING W. ..... Medical Arts Bldg., OALR
HOWARD, W. H. ..... Medical Arts Bldg., OALR
LITCHFIELD, J. T. ..... Medical Arts Bldg., OALR
LOOMIS, E. A. ..... Medical Arts Bldg., OALR
MACNIE, J. S. ..... 74 S. 11th St., OALR
MATTHEWS, JUSTUS ..... Medical Arts Bldg., LR
MORTON, HOWARD McI. ..... Insurance Exchange Bldg., Op.
NEWHART, HORACE ..... Medical Arts Bldg., ALR
OBERG, C. M. ..... Syndicate Bldg., OALR
PATTERSON, W. E. ..... 74 S. 11th St., OALR
PHELPS, KENNETH A. ..... 730 La Salle Bldg., OALR
PRATT, FRED J. ..... Medical Arts Bldg., OALR
PRATT, JOHN A. ..... Medical Arts Bldg., OALR
REYNOLDS, J. S. ..... Medical Arts Bldg., OALR
RUCKER, CHAS. W. ..... Medical Arts Bldg., Op.
SCHWARTZ, VIRGIL JOSEPH ..... Medical Arts Bldg., ALR
SMITH, ARTHUR E. ..... Donaldson Bldg., OALR
STROUT, E. S. ..... 74 S. 11th St., OALR
STROUT, G. E. ..... 1009 Nicollet Ave., OALR
WATSON, JAMES A. ..... 74 S. 11th St., OALR
WOOD, DOUGLAS R. ..... Medical Arts Bldg., OALR
WRIGHT, C. DARCY ..... Medical Arts Bldg., OALR

NEW ULM

REINEKE, GEORGE F. ..... Box 137, OALR

ROCHESTER

ANDERSON, CARL M. ..... Mayo Clinic, ALR
BENEDICT, WILLIAM L. ..... Mayo Clinic, Op.
FIGI, F. A. ..... Mayo Clinic, LR
HEMPSTEAD, BERT E. ..... Mayo Clinic, ALR
LILLIE, HAROLD I. ..... Mayo Clinic, ALR
LILLIE, W. IVAN ..... Mayo Clinic, Op.
NEW, GORDON B. ..... Mayo Clinic, L
PRANGEN, AVERY DE H. ..... Mayo Clinic, Op.
STARK, WM. B. ..... Mayo Clinic, ALR
WAGENER, HENRY P. ..... Mayo Clinic, Op.

ST. CLOUD

WENNER, W. T. ..... St. Mary's Bldg., Op.

ST. PAUL

BERRISFORD, PAUL D.....Lowry Bldg., OALR
BINGER, H. E.....Lowry Medical Bldg., OALR
BOECKMANN, EGIL.....Medical Arts Bldg., OALR
BRAY, E. R.....Lowry Medical Arts Bldg., OALR
BROWN, JOHN C.....Medical Arts Bldg., OALR
BURCH, FRANK E.....754 Linwood Pl., Op.
CONNOR, CHAS. E.....Miller Clinic, ALR
FOGARTY, CHAS. W...St. Paul Clinic Bldg., St. Peter & 5th Sts., OALR
FULTON, JOHN F.....833 Lowry Bldg., OALR
GRANT, H. W.....Hamm Bldg., Op.
LARSEN, CARL L.....1027 Lowry Medical Bldg., OALR
LEAVENWORTH, R. O.....Lowry Medical Bldg., Op.
LEWIS, WILLIAM W.....836 Lowry Bldg., OALR
MALONEY, THOMAS J.....1032 Lowry Bldg., OALR
NELSON, LOUIS A.....830 Lowry Bldg., Op.
ROTHSCHILD, HAROLD J.....239 Lowry Bldg., OALR
SHELLMAN, J. L.....Lowry Medical Arts Bldg., OALR
WHEELER, MERRITT W.....1027 Lowry Bldg., OALR
WOLD, K. C.....Lowry Medical Arts Bldg., OALR

WINONA

RISSER, EDWARD D.....310 Choate Bldg., OALR
WALKER, GEORGE H.....172 Main St., OALR

WORTHINGTON

STANLEY, C. R.....ALR

MISSISSIPPI

GREENVILLE

MONTGOMERY, DANIEL C.....Greenley Bldg., ALR

GULFPORT

MCWILLIAMS, CHAS. A.....G. & S. I. Bldg., ALR

JACKSON

ADKINS, GEORGE E.....Jackson Infirmary, ALR
HARRIS, WM. ROBIN.....Lamar Life Bldg., ALR

NATCHEZ

GAUDET, LUCIAN S.....405 Franklin St., OALR

OXFORD

GUYTON, B. S.....512 Van Buren, OALR

VICKSBURG

BELL, M. H.....First National Bank Bldg., Op.
EDWARDS, C. J.....First National Bank Bldg., OALR
HOWARD, E. F.....First National Bank Bldg., ALR
JONES, EDLEY H.....First National Bank Bldg., OALR

MISSOURI

CARTHAGE

POST, WINFRED B. ..... First National Bank Bldg., OALR
POWERS, EVERETT ..... Central National Bank Bldg., OALR

COLUMBIA

SNEED, CARL M. ..... 506 W. Broadway, OALR

KANSAS CITY

ALLEN, CHAS. E. ..... 712 Waldheim Bldg., OALR
ALTRINGER, A. N. ..... 710 Rialto Bldg., ALR
BEAL, HOMER A. ..... 1002 Argyle Bldg., ALR
BEIL, J. W. ..... Argyle Bldg., OALR
BELLOWS, GEORGE E. ..... 1500 Professional Bldg., Op.
BLAKESLEY, T. S. ..... Professional Bldg., OALR
CURDY, R. J. ..... 906 Grand Ave., Op.
CURRAN, E. J. ..... 1805 Fed. Res. Bank Bldg., Op.
DAVIS, HARRY B. ..... Waldheim Bldg., OALR
DIXON, OTTO J. ..... 902 Medical Arts Bldg., ALR
EUBANK, AMBROSE E. ..... 636 Argyle Bldg., OALR
FOSTER, HAL ..... 1426 Professional Bldg., ALR
GILLILAND, O. S. ..... Professional Bldg., ALR
HEDRICK, HAROLD B. ..... 910 Rialto Bldg., ALR
HOWARD, J. W. ..... Rialto Bldg., OALR
KEITH, W. E. ..... Professional Bldg., OALR
LANING, J. H. ..... 907 Rialto Bldg., ALR
LEMOINE, A. N. ..... Rialto Bldg., Op.
LICHTENBERG, J. S. ..... Rialto Bldg., Op.
LORIE, ALVIN J. ..... Commerce Bldg., ALR
LUX, PAUL ..... 409 Commerce Bldg., ALR
MCALESTER, ANDREW W., JR. ..... Rialto Bldg., Op.
MCCARTY, VIRGIL W. ..... Professional Bldg., OALR
MCKEE, JOSEPH W. ..... 1222 Rialto Bldg., Op.
MILLER, HUGH ..... Chambers Bldg., OALR
MOTT, JOHN S. ..... 214 Argyle Bldg., OALR
MYERS, JOHN L. ..... 815 Shukert Bldg., OALR
PAINTER, A. M. ..... Professional Bldg., ALR
ROBERTS, S. E. ..... Professional Bldg., ALR
SCHUTZ, W. H. ..... Bryant Bldg., OALR
SHOEMAKER, FERDINAND
U. S. Vet. Hosp., No. 67, 27th and Paseo Sts., OALR
SHUMATE, D. L. ..... 500 Argyle Bldg., OALR
SIMPSON, M. B. ..... Rialto Bldg., ALR
THOMASON, H. E. ..... Rialto Bldg., ALR
TEALL, RAYMOND E. ..... Professional Bldg., ALR
YAZEL, HERMAN E. ..... 733 Rialto Bldg., OALR

ST. JOSEPH

FORGRAVE, L. ROBERT.....204 Logan Bldg., OALR
MINTON, WM. H.....First State Bank Bldg., OALR
PROUD, WILLARD C.....Phys. and Surg. Bldg., OALR

ST. LOUIS

ALDEN, ARTHUR M.....537 Frisco Bldg., ALR
ALVIS, BENNETT Y.....900 Carleton Bldg., Op.
ARBUCKLE, MILLARD F.....1011 Beaumont Med. Bldg., ALR
BARDENHEIER, F. G. A.....Frisco Bldg., ALR
BLACK, W. D.....Metropolitan Bldg., ALR
BOEMER, L. C.....3720 Washington Blvd., ALR
BROWN, CLYDE O.....3720 Washington Blvd., ALR
BURNS, S. S.....Missouri Theater Bldg., ALR
CALHOUN, JAMES GRAHAM.....Metropolitan Bldg., Op.
CHARLES, JOS. W.....3720 Washington Ave., Op.
COSTEN, J. B.....Beaumont Medical Bldg., ALR
DAVIS, CHARLES L.....3720 Washington Blvd., ALR
DEAN, L. W.....Medical School, Washington Univ., ALR
DOBBS, OTTO ROSS.....2435 N. Grand Blvd., ALR
DONNELL, N. R.....Metropolitan Bldg., Op.
DYER, C. P.....408 Humboldt Bldg., Op.
EBER, CARL THEODORE.....1006 Carleton Bldg., Op.
EIMER, CHARLES EUGENE.....316 Lister Bldg., ALR
FERRIS, DAVID P.....1000 Carleton Bldg., ALR
FINEBERG, MAXWELL.....3858 Westminster Place, ALR
FREIMUTH, L. E.....University Club Bldg., ALR
GLICK, HARRY N.....3115 S. Grand Blvd., ALR
GOLDSTEIN, M. A.....3858 Westminster Place, ALR
GREEN, JOHN.....3720 Washington Blvd., Op.
GREENE, M. L.....Missouri Theater Bldg., Op.
GUGGENHEIM, L. K.....Carleton Bldg., ALR
GUNDELACH, C. A.....825 University Club Bldg., ALR
HANSEL, FRENCH KELLER.....Beaumont Medical Bldg., ALR
HARDESTY, J. F.....706 Missouri Bldg., Op.
HARDY, W. F.....Humboldt Bldg., Op.
HOBART, CARL.....Metropolitan Bldg., Op.
HOOSS, C. H. A.....3115 S. Grand Blvd., Op.
HOURN, GEORGE E.....511 University Club Bldg., ALR
HOWARD, HARVEY J.....School of Med., Washington Univ., Op.
JACOBS, MAX W.....Missouri Theater Bldg., Op.
JONES, VINCENT L.....3511 Washington Ave., Op.
KELLEY, ISAAC D., JR.....3720 Washington Blvd., ALR
LAMB, HARVEY D.....826 Metropolitan Bldg., Op.
LEAVY, C. A.....Metropolitan Bldg., ALR
LUEDDE, W. H.....Metropolitan Bldg., Op.
LYMAN, HARRY W.....Carleton Bldg., ALR
MCMAHON, BERNARD JOHN.....Missouri Theater Bldg., ALR

MASON, ROY EARL.....Frisco Bldg., Op.
MILLER, HERMAN B.....810 Metropolitan Bldg., ALR
MILLER, H. ED.....728 Missouri Bldg., ALR
MYERS, EUGENE LEE.....Wall Bldg., ALR
NEMOURS, PAUL ROLAND.....3852 Greer Ave., ALR
PAYNE, R. J.....University Club Bldg., ALR
PELZ, MORT D.....Missouri Bldg., ALR
PFINGSTEN, C. F.....6370 Waterman Ave., ALR
POOS, GROVER H.....Univ. Club Bldg., OALR
POST, LAWRENCE T.....Metropolitan Bldg., Op.
POST, M. HAYWARD.....Metropolitan Bldg., Op.
POTTER, ALLEN B.....3725 S. Kingshighway, ALR
PROETZ, A. W.....Beaumont Bldg., ALR
REIM, HUGO.....417 Metropolitan Bldg., Op.
RYAN, L. M.....Wall Bldg., ALR
SAUER, W. E.....Beaumont Medical Bldg., ALR
SCHLUER, E. P.....Metropolitan Bldg., ALR
SCHOLZ, R. P.....Metropolitan Bldg., ALR
SCHWARTZ, FREDERICK O.....Metropolitan Bldg., Op.
SENSENEY, EUGENE T.....316 Lister Bldg., ALR
SHAHAN, W. E.....Metropolitan Bldg., Op.
SHOEMAKER, W. A.....1006 Carleton Bldg., Op.
SIMON, FREDERICK C.....914 Arcade Bldg., ALR
SMIT, WM. M.....719 Missouri Theater Bldg., ALR
TOOKER, C. W.....Carleton Bldg., Op.
VAN METER, EUGENE R.....4473 Washington Ave., ALR
WESTLAKE, SAMUEL B.....Beaumont Med. Bldg., ALR
WIENER, MEYER.....900 Carleton Bldg., Op.
WOOD, V. VISSCHER.....Beaumont Medical Bldg., ALR
WOODRUFF, FREDERICK E.....Metropolitan Bldg., Op.

SEDALIA

LOVE, JOHN G.....Ilgenfritz Bldg., OALR
TISWORTH, GUY.....111 W. 4th St., OALR

SPRINGFIELD

BAILEY, HAROLD.....840 Landers Bldg., OALR
COFFELT, THEODORE A.....1240 Benton Ave., OALR
KLINGNER, THOS. O.....318 Landers Bldg., OALR
SMITH, CARLIE SOUTER.....214 N. Jefferson Ave., OALR

TRENTON

AMBROSE, ELMER C.....910½ Main St., OALR

MONTANA

ANACONDA

DUNLAP, LAWRENCE G.....101 Main St., OALR

BUTTE

DONOVAN, JOHN A. ..... 507 Phoenix Bldg., OALR
MORSE, A. W. ..... 507 Phoenix Bldg., OALR
POTTER, PETER ..... Motor Clinic Bldg., OALR

GREAT FALLS

COULTER, CHAS. F. ..... First Nat'l Bank Bldg., OALR

HAVRE

BRIGGS, F. W. ..... OALR

ROUNDUP

LEWIS, GEORGE A. ..... Wall Bldg., OALR

NEBRASKA

BEATRICE

TUCKER, JAMES C. ..... Arcade Bldg., OALR

COLUMBUS

CAMPBELL, C. H. ..... Col. State Bank Bldg., OALR
KOEBBE, EZRA EDWIN ..... ALR

GRAND ISLAND

RIGG, JAMES P. ..... Grand Island Clinic, ALR

HASTINGS

MARTIN, ROY DAVID ..... Foote Bldg., ALR

KEARNEY

FOX, CHAS. H. ..... 5 West 21st St., OALR

LINCOLN

BLACK, PAUL ..... Stuart Bldg., Op.
BROOKS, EARL B. ..... 939 Stuart Bldg., OALR
CURTIS, W. L. ..... 702 Sharp Bldg., OALR
FURGASON, A. P. ..... Richards Block, OALR
HOMPES, J. J. ..... Sharp Bldg., OALR
SANDERSON, DAVID D. ..... Stuart Bldg., OALR
THOMAS, JAMES W. ..... Sharp Bldg., OALR
ZEMER, S. G. ..... 950 Stuart Bldg., OALR

NEBRASKA CITY

STONECYPHER, D. D. ..... 107 S. 9th St., ALR

NORFOLK

GADBOIS, ARTHUR E. ..... 421 Norfolk Ave., OALR
NELSON, LLOYD L. ..... Fleming Bldg., OALR

OMAHA

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CALLFAS, W. F. ..... 1620 Medical Arts Bldg., ALR

DAVIS, J. CALVIN.....425 Aquila Court, OALR
FAIRCHILD, NORA M.....Medical Arts Bldg., Op.
HANEY, WM. P.....1500 Medical Arts Bldg., OALR
JUDD, J. H.....Medical Arts Bldg., Op.
KNODE, A. R.....708 Medical Arts Bldg., ALR
KULLY, B. M.....Medical Arts Bldg., ALR
OWEN, F. S.....Medical Arts Bldg., OALR
POTTER, GEORGE B.....Medical Arts Bldg., OALR
POTTS, J. B.....Medical Arts Bldg., ALR
ROMONEK, PHILIP L.....Medical Arts Bldg., OALR
RUBENDALL, C.....Medical Arts Bldg., OALR
SWAB, CHARLES M.....Medical Arts Bldg., Op.
UREN, C. T.....537 City National Bank Bldg., ALR
WHERRY, W. P.....1500 Medical Arts Bldg., OALR

NEVADA

RENO

FULLER, JOHN ANDRE.....First Nat'l Bank Bldg., OALR

NEW HAMPSHIRE

NASHUA

NUTTER, C. F.....16 Amherst St., Op.

NEW JERSEY

ASBURY PARK

FISHER, JAMES A.....Jersey Central Bldg., OALR
UPHAM, HELEN F.....305 Third Ave., OALR

ATLANTIC CITY

CHARLTON, C. COULTER.....124 S. Illinois Ave., ALR
MCGIVERN, C. S.....101 S. Indiana Ave., ALR
OLMSTEAD, WM. DE F.....1920 Pacific Ave., Op.

BLOOMFIELD

MOORE, JOHN D.....6 Washington St., OALR

CAMDEN

KLINE, ORAM R.....414 Cooper St., ALR

EAST ORANGE

BUVINGER, CHARLES W.....50 Washington St., OALR

HACKENSACK

GREENFIELD, WILLIAM JOHN.....50 Anderson St., ALR

JERSEY CITY

PYLE, WALLACE.....15 Exchange Pl., OALR

LONG BRANCH

CAMPBELL, W. K.....96 Third Ave., OALR

NEWARK

CARDWELL, EDGAR PARMELE.....15 Fulton St., ALR
CHATTIN, J. FRANKLIN.....671 Broad St., Op.
EAGLETON, WELLS P.....15 Lombardy St., ALR
HAHN, WM. H.....15 Lombardy St., Op.
HUGHES, L. W.....965 Broad St., OALR
HURFF, J. W.....86 Washington St., OALR
KEIM, WILLIAM F.....25 Roseville Ave., ALR
O'CONNOR, D. F.....671 Broad St., OALR
ORTON, HENRY B.....24 Commerce St., ALR
QUINBY, W. O'G.....14 James St., OALR
SHERMAN, ELBERT S.....671 Broad St., Op.
WOOD, EARL LEROY.....192 Roseville Ave., ALR
ZEHNDER, A. CHARLES.....188 Roseville Ave., Op.

ORANGE

EMERSON, LINN.....Metropolitan Bldg., OALR

PLAINFIELD

CHILDERS, ROBERT JEFFERSON.....604 Park Ave., ALR
VAN HORN, A. F.....514 Central Ave., ALR

SUMMIT.

BURRITT, NORMAN W.....30 Beachwood Road, ALR

TRENTON

ADAMS, C. F.....34 W. State St., OALR
CRANE, J. WELLINGTON.....128 Third Ave., OALR

UNION CITY

SACCO, A.....440 New York Ave., OALR

NEW MEXICO

ALBUQUERQUE

BREHMER, HARRISON L.....First Nat'l Bank Bldg., OALR

FORT BAYARD

GILL, ARTHUR E.....U. S. Veterans' Hospital, OALR

NEW YORK

ALBANY

BEDELL, ARTHUR J.....344 State St., Op.
DOWLING, J. IVIMEY.....116 Washington Ave., OALR
HINMAN, E. E.....146 Washington Ave., ALR
JUDGE, HARRY V.....46 Willett St., Op.
TEBBUTT, H. K.....240 State St., ALR

BINGHAMTON

KINNE, SANFORD HALSEY.....73 Main St., ALR
RANDALL, HIRAM.....81 Main St., ALR
WATSON, H. D.....151 Front St., OALR

BROOKLYN

AGAN, WM. B.....510 Third St., Op.
ARROWSMITH, HUBERT.....11 Schermerhorn St., ALR
CUTINO, R. M.....165 Clinton St., Op.
DE YOANNA, ALFRED A.....111 Pierrepont St., ALR
EBELING, WM. B.....54 Greene Ave., Op.
EVANS, J. N.....23 Schermerhorn St., Op.
GOLDING, JOSEPH E.....170 Hancock St., Op.
GREENFIELD, SAMUEL D.....169 New York Ave., ALR
HANCOCK, J. C.....135 Cambridge Pl., Op.
HARGITT, CHARLES A.....17 Schermerhorn St., Op.
KRIMSKY, JOSEPH.....263 Eastern Parkway, OALR
KRUSKAL, I. D.....863 Prospect Pl., Op.
LLOYD, RALPH I.....14 Eighth Ave., Op.
LOMBARDO, MELCHIORE.....142 Joralemon St., Op.
MENGER, LOUIS CHARLES.....202 Weirfield St., ALR
O'BRIEN, G. R.....307 Sterling Place, ALR
ROGERS, ROBERT M.....680 St. Johns Pl., Op.
THOMASHEFSKY, M.....135 Eastern Parkway, ALR
WEETH, CHAS. R.....37 Remsen St., ALR

BUFFALO

ANDREWS, H. D.....33 Allen St., Op.
BENNETT, ARTHUR G.....150 N. Pearl St., Op.
BEYER, LOUIS J.....449 Delaware Ave., ALR
BLAAUW, E. E.....190 Ashland Ave., Op.
BOZER, HERRMANN EUGENE.....468 Delaware Ave., ALR
BROWN, C. M.....510 Delaware Ave., ALR
COTT, C. C.....104 Dorchester Road, ALR
COWPER, H. W.....543 Franklin St., Op.
EDSON, R. A.....498 Delaware Ave., Op.
FAIRBAIRN, JOHN F.....925 Delaware Ave., ALR
FLAGG, J. D.....158 Anderson Place, Op.A
GLOSSER, HERBERT H.....448 Franklin St., Op.
HEALY, JOHN F.....503 Delaware Ave., ALR
KOENIG, IVAN J.....40 North St., Op.
LE WIN, THURBER.....112 Linwood Ave., Op.
LEWIS, F. PARK.....454 Franklin St., Op.
MCKEE, OTTO S.....83 Bryant St., ALR
MARCH, CLARA A.....465 Ashland Ave., Op.
PHILLIPS, WM. LINTON.....131 Linwood Ave., Op.
RENNER, W. S.....341 Linwood Ave., ALR
WEED, HARRY M.....196 Linwood Ave., Op.
WURTZ, W. J. M.....333 Linwood Ave., ALR

CORTLAND

CUTLER, FRANKLIN E.....R. D. No. 2, ALR
HIGGINS, R. P.....20 Court St., OALR
WHITE, O. E.....28 N. Main St., ALR

DUNKIRK

JACKLE, ARTHUR F.....63 E. 4th St., Op.

FLUSHING

GOODE, EDWARD ALLEN.....239 Sanford Ave., ALR

GENEVA

SPENGLER, JOHN ARTHUR.....423 S. Main St., Op.

GLENS FALLS

FIELDING, F. G.....70 Ridge St., ALR

JAMAICA

BENDER, M. S.....11-150 St., ALR

JAMESTOWN

REGER, H. S.....Physicians Bldg., OALR

LOCKPORT

RINGUEBERG, E. N. S.....13 Main St., Op.

MIDDLETOWN

HICKS, WM. J.....15 King St., ALR

MT. VERNON

KEEFE, WM. JOHN.....110 E. Sidney Ave., ALR
THOMSON, J. J.....531 E. Lincoln Ave., ALR

NEW ROCHELLE

BECK, A. L.....421 Huguenot St., OALR
WOODRUFF, WALTER STUART.....421 Huguenot St., ALR

NEW YORK CITY

ALMOUR, RALPH W.....51 W. 73rd St., ALR
AUERBACH, JULIUS.....1016 Fifth Ave., ALR
BALLIN, M. J.....20 E. 76th St ALR
BASSEN, ED. J.....698 Madison Ave., Op.
BEACH, B. S.....140 W. 86th St., Op.
BENDER, M. S.....30 E. 40th St., ALR
BERENS, CONRAD.....35 E. 70th St., Op.
BRUCE, G. M.....180 Fort Washington Ave., Op.
BRUDER, JOSEPH.....57 W. 57th St., OALR
CARTER, W. W.....2 E. 54th St., ALR
CARY, ED. F.....171 W. 57th St., ALR
COHEN, MARTIN.....1 W. 85th St., Op.
COHN, FELIX.....47 W. 87th St., ALR
COLBERT, ROBERT MARTIN.....57 W. 57th St., ALR
CORYA, HAROLD W.....30 E. 40th St., ALR
COTT, D. N.....125 Central Ave., St. George, Staten Isl., ALR
CUNNING, DANIEL S.....145 E. 54th St., OALR
DAVIDSON, MORRIS.....210 W. 70th St., Op.

DAVIS, A. EDWARD.....40 E. 61st St., Op.
DAVIS, G. E.....1060 Fifth Ave., OALR
DEPPING, CHAS. WM.....17 W. 54th St., ALR
DIXON, G. S.....1150 Fifth Ave., R
DLUGASCH, LOUIS.....110 E. 36th St., ALR
DOHERTY, W. B.....150 W. 55th St., Op.
DUNNINGTON, J. H.....30 W. 59 St., Op.
ELLIS, Z. H.....58 East 65th St., Op.
FERNLUND, GEORGE BYRON
Hillside Ave. and 118th St., Richmond Hill, OALR
FOWLER, ROBERT HENRY.....153 E. 62nd St., ALR
FOWLKES, JOHN WINSTON.....30 W. 59th St., ALR
FRANKEL, EDWARD, JR.....152 W. 58th St., ALR
GATEWOOD, W. L.....330 Park Ave., ALR
GOLDMAN, A. MILTON.....121 E. 60th St., Op.
GOLDMAN, H. G.....57 W. 58th St., ALR
GOLDSTEIN, ISADORE.....1060 Park Ave., Op.
GOTTLIEB, MARK J.....2 W. 87th St., ALR
GRESSER, EDWARD BELLAMY.....39 Fifth Ave., Op.
GRUSHLOW, ISRAEL.....271 Central Park, W., ALR
GUTTMAN, J.....1235 Park Ave., OALR
HALLETT, G. DEWAYNE.....274 W. 86th St., Op.
HANCOCK, J. C.....2 Rector St., Op.
HARDY, LEGRAND H.....30 E. 40th St., Op.
HARRIS, THOMAS JEFFERSON.....104 E. 40th St., ALR
HARTSHORNE, ISAAC.....30 W. 59th St., Op.
HASKIN, W. H.....57 W. 57th St., ALR
HAYS, HAROLD M.....64 E. 58th St., ALR
HETRICK, L. E.....40 E. 61st St., ALR
HORN, WALTER L.....1040 Park Ave., ALR
HURD, L. M.....39 E. 50th St., ALR
HUGHES, WENDELL LOCHEAD.....20 E. 53 St., Op.
HUNT, WESTLEY MARSHALL.....33 E. 68th St., ALR
HUTCHISON, ALBERT T.....30 W. 59th St., ALR
IMPERATORI, C. J.....17 E. 38th St., ALR
INGERMAN, S. M.....418 Central Park, W., OALR
JACOBS, S. M.....1018 E. 163rd St., OALR
JARECKY, HERMAN.....168 W. 86th St., OALR
JOHNSON, THOMAS HOLLAND.....30 W. 59th St., Op.
JONES, MARVIN F.....121 E. 60th St., ALR
JOSEPHSON, EMANUEL M.....993 Park Ave., ALR
JUDD, H. B.....40 E. 61st St., ALR
KELLY, JOSEPH DOMINIC.....114 E. 54th St., OALR
KEY, BEN WITT.....100 W. 59th St., Op.
KING, J. J.....30 E. 40th St., ALR
KIRBY, D. B.....30 W. 59th St., Op.
KNAPP, ARNOLD.....10 E. 54th St., Op.
KOPETZKY, S. J.....51 W. 73rd St., ALR
KRUG, ERNEST F.....12 W. 44th St., Op.

La Vigne, A. A. ..... 471 Park Ave., OALR
Lubman, Max ..... 17 E. 96th St., ALR
McCoy, John ..... 730 Fifth Ave., ALR
McDannald, C. E. ..... 100 W. 59th St., Op.
McDonald, Dennis J. ..... 17 E. 38th St., ALR
May, Chas. H. ..... 698 Madison Ave., Op.
Maybaum, J. L. ..... 1079 Park Ave., ALR
Mayer, Emil ..... 320 W. 89th St., ALR
Meierhof, E. L. ..... 12 E. 86th St., OALR
Miller, John ..... 121 E. 60th St., ALR
Miller, Joseph William ..... 305 E. 17th St., ALR
Minsky, Henry ..... 1 West 86th St., Op.
Mittendorf, A. D. ..... 339 Park Ave., Op.
Mittendorf, W. Keith ..... 115 E. 53rd St., Op.
Morrison, Wm. Wallace ..... 39 E. 50th St., ALR
Myerson, M. C. ..... 12 E. 86th St., ALR
Nisselson, Max ..... 754 Beck St., ALR
Northcott, Thomas A. ..... 17 E. 38th St., Op.
Oast, Samuel P. ..... 30 E. 40th St., Op.
Palmer, Arthur ..... 667 Madison Ave., ALR
Peterson, R. A. ..... 150 Fifth Ave., OALR
Reese, Algernon B. ..... 50 W. 52nd St., Op.
Rosenbaum, Morris ..... 604 E. 5th St., Op.
Samuels, Bernard ..... 57 W. 57th St., Op.
Scal, Joseph Coleman ..... 44 W. 74th St., ALR
Schiller, A. N. ..... 101 Central Park, West, ALR
Schlivek, Kaufman ..... 1016 Fifth Ave., Op.
Schwartz, A. A. ..... 941 Park Ave., ALR
Somberg, J. S. ..... 470 Park Ave., Op.
Stark, Jesse B. ..... 45 Park Ave., Op.
Sturges, Leigh F. ..... 121 E. 60th St., ALR
Weeks, Webb William ..... 20 E. 53rd St., Op.
Wexler, David ..... 55 E. 86th St., Op.
Wheeler, J. M. ..... 30 W. 59th St., Op.
Wiener, Alfred ..... 550 Park Ave., OALR
Yankauer, Sidney ..... 121 E. 60th St., ALR

NIAGARA FALLS

Price, Norman W. ..... 445 Third St., OALR
Rooker, A. M. ..... 203 Gluck Bldg., ALR

NYACK

Toms, S. W. S. ..... 120 S. Broadway, OALR

OLEAN

Tindolph, L. W. ..... First Nat'l Bank Bldg., ALR

ONEIDA

Crockett, R. L. ..... 302 Main St., Op.

PLATTSBURG

SCHNEIDER, ANTON S.....56 Court St., Op.A

POUGHKEEPSIE

KRIEGER, WM. A.....35 Market St., OALR

ROCHESTER

BARBER, F.....75 S. Fitzhugh St., Op.
BARLOW, ROY A.....Hiram-Sibley Bldg., ALR
CARROLL, GEORGE G.....5 North Goodman St., OALR
CLARK, L. H.....337 Monroe Ave., OALR
GIPNER, J. F.....53 S. Fitzhugh St., Op.
JONES, L. W.....16 N. Goodman St., OALR
KENNEDY, ELDRED WESTON.....Gas and Electric Bldg., Op.
LERNER, MACY LEVI.....332 Park Ave., Op.
MCDOWELL, N. D.....275 Alexander St., OALR
MORRIS, A. G.....277 Alexander St., ALR
NASH, C. STEWART.....26 S. Goodman St., ALR
SHAPERO, I. M.....289 Alexander St., OALR
SNELL, ALBERT C.....53 S. Fitzhugh St., Op.

SYRACUSE

BRITTEN, G. S.....Medical Arts Bldg., ALR
BROWN, MORTIMER G.....Medical Arts Bldg., ALR
BRUST, H. O.....820 S. Crouse Ave., ALR
CAVE, LINUS S.....221 Physicians Bldg., ALR
FOWLER, S. R.....University Block, ALR
GILLETTE, D. F.....State Tower Bldg., Op.
HALL, EDWIN P.....State Tower Bldg., ALR
HOOPLE, GORDON D.....Medical Arts Bldg., ALR
JOY, HAROLD HENRY.....504 State Tower Bldg., Op.
KAUFFMAN, A. B.....Chimes Bldg., ALR
KLINE, H. G.....State Tower Bldg., ALR
MARLOW, SEARLE B.....1004 State Tower Bldg., Op.
MOORE, ROY S.....Medical Arts Bldg., ALR
RUBENSTEIN, A. H.....Medical Arts Bldg., ALR
WEBSTER, F. R.....720 State Tower Bldg., Op.

TROY

SULZMAN, FRANK MALCOLM.....1831 Fifth Ave., OALR

UTICA

BEATTIE, WILLIAM H.....258 Genesee St., OALR

WATERTOWN

ATKINSON, WALTER S.....168 Sterling St., Op.
HENDERSON, L. E.....168 Sterling St., Op.

WHITE PLAINS

MORIARTY, RICHARD W.....Medical Center, OALR

WOODHAVEN

FROTHINGHAM, EDWARD ..... 8644 94th St., OALR

NORTH CAROLINA

ASHEVILLE

BRIGGS, H. H. ..... 101 Haywood St., OALR
GREENE, JOSEPH BERRY ..... 305 Haywood Bldg., ALR

CHARLOTTE

MATHESON, J. P. ..... 6 W. 7th St., ALR
PEELER, C. N. ..... 6 W. 7th St., OALR
SLOAN, HENRY L. ..... 6 W. 7th St., Op.
WHISNANT, A. M. ..... 517 Professional Bldg., OALR

GREENSBORO

REAVES, CHAS. R. ..... Reaves Infirmary, 117 W. Sycamore St., OALR
REAVES, W. P. ..... Reaves Infirmary, 117 W. Sycamore St., OALR

KINSTON

PEERY, VANCE PRICE ..... ALR

MOREHEAD CITY

JOHNSON, OTIS H. ..... OALR

NEW BERN

DANIELS, R. L. ..... 48 East Front St., OALR

RALEIGH

HICKS, VONNIE MONROE ..... Professional Bldg., Op.
WRIGHT, J. B. ..... Professional Bldg., ALR

WILMINGTON

MURPHY, J. G. ..... Murchison Bldg., OALR

WINSTON-SALEM

DAVIS, THOMAS W. ..... 210 O'Hanlon Bldg., OALR

NORTH DAKOTA

BISMARCK

CONSTANS, GEORGE MAURICE ..... Quain and Ramstad Clinic, Op.

FARGO

RINDLAUB, ELIZABETH P. ..... 620 Front St., OALR

MINOT

MCCANNEL, A. D. ..... 33 Central Ave. W., OALR

OHIO

AKRON

BROWN, L. E. ..... Second Nat'l Bank Bldg., OALR
MOORE, T. K. ..... Second Nat'l Bank Bldg., OALR
STEVENSON, D. W. ..... 165 E. Market St., OALR

ALLIANCE

KING, GEORGE L., JR. ..... 537 Market St., Op.
KING, G. L., SR. ..... 537 E. Market St., OALR

BELLEFONTAINE

BUTLER, ROBERT H. ..... 112 W. Columbus Ave., OALR
HARBERT, JOHN P. ..... 135 N. Main St., OALR

CANTON

HOFFMANN, JOHN N. ..... 309 Brant Bldg., ALR
SCHILD, E. H. ..... Brant Bldg., Op.
TOOT, JOSEPH F. ..... 923 First Nat'l Bank Bldg., Op.

CINCINNATI

ABRAHAMSON, IRA A. ..... 605 Traction Bldg., Op.
ASBURY, MARY K. ..... 3549 Holly Lane, Op.
BEYER, A. G. ..... Doctors Bldg., OALR
BROWN, ALBERT LOUIS ..... 2600 Union Central Bldg., Op.
GOODYEAR, H. M. ..... 556 Doctors Bldg., ALR
HANS, C. L. ..... Provident Bank Bldg., OALR
HARRIS, W. C. ..... Groton Bldg., ALR
HOFLING, CHARLES A. ..... 707 Race St., Op.
IGLAUER, SAMUEL ..... 707 Race St., ALR
JONES, C. C. ..... 19 Garfield Place, ALR
KING, CLARENCE ..... Union Central Bldg., Op.
KING, ED. ..... 1002 Carew Tower, ALR
LYLE, DONALD J. ..... 19 W. 7th St., OALR
MITHOEFER, W. ..... Doctors Bldg., ALR
PHINNEY, F. D. ..... 22 W. 7th St., OALR
RAY, VICTOR ..... 19 W. 7th St., Op.
REID, H. W. ..... 19 Garfield Place, Op.
SIEGEL, FRANCIS X. ..... 901 Union Central Life Bldg., OALR
STANBERRY, HENRY ..... 556 Doctors Bldg., Op.
STEWART, T. M. ..... 901 Union Trust Bldg., OALR
STITT, H. L. ..... 656 Doctors Bldg., OALR
TANGEMAN, H. F. ..... 606 Union Central Life Bldg., ALR
THOMPSON, E. H. ..... 8 W. 9th St., OALR
URNER, M. H. ..... 2600 Union Central Bldg., OALR
VAIL, H. H. ..... 24 E. 8th St., ALR

CLEVELAND

ABBOTT, W. J. ..... 8314 Euclid Ave., OALR
BRUNER, ABRAM BRENEMAN ..... 1214 Guardian Bldg., OALR
BRUNER, WM. E. ..... Guardian Bldg., Op.
BURKE, T. A. ..... Rose Bldg., OALR
COGAN, J. E. ..... 707 Rose Bldg., OALR
DIXON, FRED WILLIS ..... 1029 Rose Bldg., ALR
FORSYTHE, S. T. ..... Rose Bldg., OALR
KENDALL, M. R. ..... Osborn Bldg., ALR
LARGE, S. H. ..... Hanna Bldg., ALR

LENKER, JOHN N. ..... 1020 Huron Road, ALR
McDONALD, C. L. ..... 964 Rose Bldg., OALR
METZ, ROY B. ..... 1214 Guardian Bldg., Op.
METZENBAUM, MYRON ..... Rose Bldg., ALR
MONSON, S. H. ..... 1322 B. F. Keith Bldg., OALR
MOORE, PAUL G. ..... Medical Arts Bldg., Op.
MOTTO, MICHAEL PAUL ..... 550 Rose Bldg., Op.
MULLIN, WM. V. ..... Cleveland Clinic, Euclid Ave. at 93rd St., ALR
MUSSUN, W. G. ..... Hanna Bldg., ALR
NELSON, CHAS. F. ..... 606 Shofield Bldg., OALR
PITKIN, C. E. ..... Osborn Bldg., ALR
PRENDERGAST, D. A. ..... Medical Arts Bldg., OALR
QUITTNER, S. S. ..... 3912 Prospect Ave., OALR
RIEGER, W. H. ..... Hanna Bldg., ALR
ROSENBERGER, HARRY CLIFFORD ..... Rose Bldg., ALR
RUEDEMANN, ALBERT DARWIN ..... Cleveland Clinic, Op.
RUST, E. G. ..... Hanna Bldg., Op.
SHACKLETON, W. E. ..... 2323 Prospect Ave., Op.
SHIRAS, HOWARD H. ..... 624 Guardian Bldg., Op.
SHIREY, O. M. ..... Osborn Bldg., ALR
SIMONDS, O. F. ..... Hanna Bldg., Op.
STOTTER, ARTHUR L. ..... 1148 Euclid Ave., OALR
TRIPP, IRA A. ..... 436 Rose Bldg., OALR
TUCKERMAN, W. C. ..... 733 Osborn Bldg., Op.
TUCKERMAN, W. H. ..... 733 Osborn Bldg., OALR
WAUGH, J. M. ..... Cleveland Clinic, Euclid Ave. at 93rd St., OALR
WEIDLEIN, IVAN F. ..... 614 Osborn Bldg., ALR

COLUMBUS

BEATTY, H. G. ..... 681 E. Broad St., ALR
BROWN, JOHN E. ..... 370 E. Town St., OALR
CLARK, I. G. ..... 188 E. State St., OALR
DAVIS, WM. C. ..... 327 E. State St., Op.
FROST, ALBERT DALBEY ..... 681 E. Broad St., Op.
HARRIS, ED. WM. ..... 681 E. Broad St., ALR
HAUER, A. M. ..... 327 E. State St., ALR
HELFRICH, ED. D. ..... 269 E. State St., ALR
MEANS, R. G. ..... 137 East State St., ALR
POSTLE, C. D. ..... 240 E. State St., ALR
PROUT, A. W. ..... 370 E. Town St., OALR
SANOR, DANIEL G., JR. ..... 206 E. State St., ALR
SILBERNAGEL, C. E. ..... 240 E. State St., OALR
STOUTENBOROUGH, WM. A. ..... 21 E. State St., OALR
SULZER, G. A. ..... 327 E. State St., OALR
THOMAS, FRANCIS W. ..... 327 E. State St., OALR
TIMBERMAN, ANDREW ..... Beggs Bldg., OALR

DAYTON

BINKLEY, ROY S. ..... Fidelity Bldg., OALR
BLACKBURN, W. J. ..... 663 Reibold Bldg., OALR

DUTROW, H. V.....1040 Fidelity Medical Bldg., OALR
FARMER, A. G.....Fidelity Medical Bldg., OALR
FOUTS, JOHN D.....910 Fidelity Medical Bldg., OALR
HARRIS, H. B.....Fidelity Medical Bldg., OALR
KILBOURNE, P. H.....Fidelity Medical Bldg., OALR
MILLETTE, J. W.....Reibold Bldg., OALR

ELYRIA

GILL, GEORGE.....Elyria Sav. and Trust Bldg., OALR

IRONTON

DUNN, O. B.....Fourth and Railroad Sts., OALR

LAKEWOOD

MERICA, F. W.....14805 Detroit Ave., ALR
MITCHELL, H. E.....15701 Detroit Ave., ALR

LIMA

STUEBER, F. G.....209 W. North St., OALR
STUEBER, PAUL J.....209 W. North St., OALR

LORAIN

MONOSMITH, O. B.....O. P. S. Bldg., OALR

MANSFIELD

GARBER, J. M.....14 N. Mulberry St., OALR
WISE, RALPH C.....News Bldg., OALR

MARIETTA

SAUER, W. W.....231 Third St., OALR

MARION

BRICKLEY, DANIEL W.....Uhler-Phillips Bldg., OALR

MASSILLON

SCOTT, MERLE E.....First Nat'l Bank Bldg., OALR

MIDDLETOWN

WILLIAMS, WALTON H.....Castell Bldg., OALR

NEWARK

HATCH, C. B.....3 W. Church St., OALR

SPRINGFIELD

EASTON, J. C.....First Nat'l Bank Bldg., OALR
HARTLEY, F. A.....302 E. High St., OALR
HOGUE, D. W.....34 W. High St., OALR
MINOR, C. L.....First Nat'l Bank Bldg., OALR

STEUBENVILLE

GOURLEY, G. F.....Sinclair Bldg. OALR

TIFFIN

PORTER, E. H. ..... 85 Madison St., OALR

TOLEDO

ALDERDYCE, WM ..... 513 Madison Ave., OALR
ALTER, F. W. ..... 219 Fifteenth St., OALR
DENMAN, IRA O. ..... 330 Richardson Bldg., OALR
KING, CHAS. R ..... Spitzer Bldg., OALR
LUKENS, CHARLES ..... 316 Michigan St., OALR
LUKENS, JOHN A. ..... 316 Michigan St., ALR
SNYDER, W. H. ..... 2001 Callingwood Ave., OALR
STEINFELD, A. L. ..... 125 Fifteenth St., OALR
WINGER, IRA B. ..... 225 Michigan St., OALR

WARREN

STIMSON, GEORGE W. ..... Packard Bldg., OALR

XENIA

MADDEN, REED ..... Allen Bldg., OALR
SHIELDS, LAWRENCE ..... Allen Bldg., ALR

YOUNGSTOWN

BEARD, HAROLD JOLLY ..... 510 Dollar Bank Bldg., OALR
HARTZELL, S. M. ..... Dollar Savings and Trust Co., OALR

ZANESVILLE

CULBERTSON, L. R. ..... Masonic Temple, OALR

OKLAHOMA

BARTLESVILLE

KISER, J. D. ..... Cent. Nat'l Bank Bldg., OALR

ENID

PIPER, ARTHUR S. ..... 610 S. Monroe, OALR

GUTHRIE

BARKER, CHAS. B. ..... 224½ W. Okla. Ave., OALR

McALESTER

DAVIS, J. E. ..... Kress Bldg., OALR

MUSKOGEE

FULLENWIDER, C. M. ..... Barnes Bldg., OALR

OKLAHOMA CITY

DIXON, W. E. ..... 503 Medical Arts Bldg., OALR
FERGUSON, E. S. ..... 902 Medical Arts Bldg., OALR
GUTHRIE, A. L. ..... Osler Bldg., OALR
MACDONALD, J. C. ..... 301 W. 12th St., ALR
McHENRY, D. D. ..... 604 Medical Arts Bldg., OALR
McHENRY, L. C. ..... 117 N. Broadway, ALR
NEWTON, L. A. ..... 609 Medical Arts Bldg., OALR

TULSA

BRASWELL, JAMES C.....Medical Arts Bldg., OALR
COOK, W. ALBERT.....Medical Arts Bldg., OALR
HARALSON, CHARLES HERNDON.....Medical Arts Bldg., Op.
ROTH, ALBERT W.....Medical Arts Bldg., OALR
SMITH, RURIC N.....Medical Arts Bldg., ALR

OREGON

BAKER

GORDON, GLENN E.....First Nat'l Bank Bldg., ALR

LA GRANDE

BOUVY, L. B.....Foley Bldg., OALR

PORTLAND

AINSLIE, GEORGE.....Selling Bldg., OALR
BOUVY, HARRY M.....800 Pittock Blk., OALR
BRUERE, GUSTAVE E.....Medical-Dental Bldg., OALR
CARRUTH, HOWARD ERNEST.....Medical Arts Bldg., OALR
DAVIS, RALPH F.....Mayer Bldg., OALR
DICKSON, JOHN F.....418 Mayer Bldg., OALR
FENTON, RALPH A.....806 Medical Arts Bldg., OALR
HENTON, G. E.....Morgan Bldg., OALR
JOHNSTON, WILSON.....611 Lovejoy St., OALR
KARKEET, ROBERT B.....1203 Selling Bldg., OALR
KIEHLE, F. A.....406 Taylor St., OALR
KISTNER, F. B.....600 Mayer Bldg., OALR
LUCAS, HAROLD R.....Medical-Dental Bldg., OALR
LUPTON, IRVING MARTIN.....Medical Arts Bldg., OALR
WEEKS, JOHN E.....360 Edgecliff Road (Retired), Op.

SALEM

BAUM, WM. W.....Bank of Commerce Bldg., ALR
CLEMENT, L. O.....Guardian Bldg., OALR

PENNSYLVANIA

ALLENTOWN

UHLER, S. MANN.....104 N. Eighth, ALR

ALTOONA

GLOVER, S. P.....1118 Twelfth Ave., OALR

BEAVER

CRUMRINE, NORMAN ROSS.....595 Third St., OALR
SCROGGS, J. J.....218 E. Third St., Op.A

BETHLEHEM

CHASE, W. D.....230 E. Broad St., ALR

BRADFORD

ASH, GARRETT G.....1 Main St., OALR

BUTLER

DOANE, L. L. ..... 357 N. Main St., OALR
HAZLETT, L. R. ..... Savings and Trust Bldg., OALR

CHARLEROI

FERMAN, JOHN W. ..... Stahlman Bldg., OALR

CHESTER

CROSS, G. H. ..... 525 Welsh St., Op.
ORR, ADRIAN V. B. ..... 525 Welsh St., ALR

CONNELLSVILLE

HARRISON, FRED H. ..... 308 First Nat'l Bank Bldg., OALR

DU BOIS

BEALS, C. WEARNE ..... 41 N. Brady St., ALR

ERIE

DENNIS, DAVID N. ..... 221 W. 9th St., Op.
DENNIS, EDWARD P. ..... 309 W. 10th St., OALR
FUST, JOHN H. E. ..... 221 W. 9th St., OALR
HEARD, C. F. ..... 813 Sassafras St., OALR
JACKSON, J. D. ..... 232 W. 8th St., OALR
RUSSELL, J. A. M. ..... 813 Sassafras St., OALR
SCHLINDWEIN, G. WM. ..... 138 W. 9th St., OALR
SHREVE, OWEN M. ..... 162 W. 8th St., Op.

FRANKLIN

JOBSON, G. B. ..... West Park and Liberty St., OALR

GREENSBURG

FAIRING, JOHN W. ..... 206 Tribune Review Bldg., OALR
MCKEE, CLAUD W. ..... Coulter Bldg., OALR

HARRISBURG

DAILEY, GILBERT L. ..... 618 N. 3rd St., OALR
FARNSLER, H. HERSHEY ..... 1438 Market St., OALR
GRACEY, GEORGE F. ..... 209 State St., OALR
PARK, J. WALTER ..... 32 N. Second St., OALR

HAZLETON

REICHE, O. C. ..... Markle Bank Bldg., OALR

JOHNSTOWN

BARKER, O. G. A. ..... 804 Johnstown Trust Bldg., Op.
GRIFFITH, HAROLD M. ..... 804 Johnstown Trust Bldg., Op.
HARRIS, C. M. ..... 604 Johnstown Trust Bldg., OALR
RUSH, C. C. ..... 907 U. S. Nat'l Bank Bldg., OALR

LEBANON

REESE, MONROE D. ..... 123 N. 8th St., OALR

MIDDLETOWN

GEORGE, HENRY W.....19 N. Union St., Op.

NEW KENSINGTON

MCCONNELL, T. E.....Logan Trust Bldg., OALR
STEIM, J. M.....First Nat'l Bank Bldg., Op.

NORRISTOWN

BEIDEMAN, JOSEPH ELLSWORTH.....Montgomery Trust Arcade, Op.
PRICE, JOHN BEADLE.....513 Swede St., ALR

PHILADELPHIA

ADLER, FRANCIS HEED.....313 S. 17th St., Op.
ALEXANDER, GEORGE J.....1930 Chestnut St., ALR
AMANO, KAGEYAS W.....502 Woodland Terrace, ALR
ANNON, W. T.....235 S. 15th St., ALR
APPLEMAN, L. F.....308 S. 16th St., Op.
BABBITT, JAMES A.....1912 Spruce St., ALR
BAESEMAN, R. WINFIELD.....928 Clinton St., ALR
BALDWIN, KATE W.....1117 Spruce St., ALR
BALENTINE, P. L.....1524 Chestnut St., Op.
BLAIR, MORTIMER, W.....N. E. Cor. 20th and Chestnut St., Op.
BOEHRINGER, W. H.....700 Central Medical Bldg., OALR
BROWN, SAMUEL HORTON.....1930 Chestnut St., Op.
BRUMM, SETH A.....Medical Arts Bldg., OALR
BURNS, L. J.....1930 Chestnut St., ALR
BUTLER, MARGARET F.....1831 Chestnut St., ALR
BUTT, MIRIAM M.....2045 Chestnut St., Op.
CARPENTER, C.....1930 Chestnut St., Op.
CLERF, LOUIS HENRY.....128 S. 10th St., ALR
COATES, GEORGE M.....1721 Pine St., ALR
COHEN, HERMAN B.....Spruce and 13th Sts., ALR
COHEN, SAMUEL.....2009 Pine St., ALR
COWAN, ALFRED.....1930 Chestnut St., Op.
CREIGHTON, WM. J.....258 S. 18th St., Op.
CROSKEY, J. W.....Medical Arts Bldg., Op.
DAVIS, J. L.....135 S. 18th St., ALR
DEICHLER, LYN W.....1737 Chestnut St., OALR
DINTENFASS, HENRY.....1305 Spruce St., ALR
EMBERY, FRANK.....1532 Overington St., ALR
ERSNER, MATTHEW S.....1915 Spruce St., ALR
FELT, C. L.....Chestnut and 20th Sts., ALR
FOX, L. WEBSTER.....303 S. 17th St., Op.
GLEASON, E. B.....2112 Pine St., ALR
GODDARD, H. M.....1531 Spruce St., ALR
GOUTERMAN, JOSEPH I.....1607 W. Diamond St., Op.
GOWEN, FRANCIS A.....2033 Pine St., ALR
GRISCOM, J. MILTON.....2213 Walnut St., Op.
HAWMAN, ERLE G.....4051 N. Broad St., Op.

HEED, C. R.....1205 Spruce St., Op.
HOLLOWAY, THOMAS B.....1819 Chestnut St., Op.
HUNTER, J. F.....1737 Chestnut St., ALR
HUNTER, R. S.....2011 Chestnut St., ALR
HUSIK, D. N.....1930 Chestnut St., ALR
JACKSON, CHEVALIER.....235 S. 15th St., L
KAUFMAN, A. SPENCER.....1923 Spruce St., ALR
KEELER, J. C.....Medical Arts Bldg., ALR
KRAUSS, FREDERICK.....1930 Chestnut St., OALR
LAESSLE, HENRY A.....The Lenox, 13th and Spruce Sts., ALR
LAWRENCE, GRANVILLE A.....Medical Arts Bldg., Op.
LEWIS, FIELDING O.....259 S. 17th St., ALR
LOFTUS, JOHN EDWARD.....1930 Chestnut St., ALR
LUKENS, ROBERT McD.....1923 Spruce St., ALR
MACKENZIE, ALICE V.....1724 Spruce St., OALR
MACKENZIE, GEORGE W.....1724 Spruce St., OALR
MANN, BENJAMIN H.....N. E. Corner 20th and Chestnut Sts., Op.
MILLER, E. B.....2008 Walnut St., Op.
MITCHELL, E. K.....710 W. Lehigh Ave., ALR
MOORE, GEORGE H.....18th and Chestnut Sts., OALR
MOORE, WILLIAM FREDERICK.....255 S. 16th St., ALR
OLSHO, S. L.....235 S. 15th St., OALR
O'REILLY, CHARLES A.....521 Medical Arts Bldg., ALR
PERSKY, ABRAM H.....1922 Spruce St., ALR
PETER, L. C.....1930 Chestnut St., Op.
PONTIUS, P. J.....1831 Chestnut St., Op.
POSEY, WM. CAMPBELL.....2049 Chestnut St., Op.
RADCLIFFE, McCLUNEY.....1930 Chestnut St., Op.
REESE, WARREN S.....230 S. 21st St., Op.
RIDPATH, ROBERT F.....N. E. Cor. 18th and Chestnut Sts., LR
ROMMEL, JOHN C.....3701 N. Broad St., Op.
RUDOLPHY, JAY BESSON.....1924 Pine St., Op.
SARTAIN, PAUL J.....2006 Walnut St., Op.
SCARLETT, HUNTER W.....230 S. 21st St., Op.
SCHNEIDEMAN, T. B.....2045 Chestnut St., Op.
SCHWEINITZ, GEORGE E. DE.....1705 Walnut St., Op.
SENER, WALTER J.....Physicians Bldg., Op.
SHANNON, C. E. G.....1930 Chestnut St., Op.
SHEMELEY, WM. G., JR.....1724 Spruce St., OALR
SHUSTER, BENJAMIN HARRISON.....1930 Chestnut St., ALR
SKILLERN, S. R., JR.....1734 Pine St., ALR
SLOANE, HENRY O.....1717 Pine St., Op.
SPAETH, EDMUND B.....1930 Chestnut St., Op.
SPENCER, W. H.....1830 S. Rittenhouse Sq., ALR
STILES, CHAS. M.....1130 Spruce St., Op.
STOUT, PHILIP S.....269 S. 19th St., ALR
TASSMAN, ISAAC S.....Medical Arts Bldg., Op.
TUCKER, GABRIEL.....326 S. 19th St., B
WAGERS, ARTHUR J.....1429 Spruce St., ALR

WEINSTEIN, M. A.....1309 Spruce St., ALR
WHELAN, G. L.....2100 Walnut St., ALR
WILLIAMS, HORACE J.....5908 Green St., Germantown, ALR
ZENTMAYER, WILLIAM....Suite 803, S. E. Cor. 20th & Chestnut, Op.

PITTSBURGH

BERNATZ, C. F.....Park Bldg., Op.
BLAIR, W. W.....121 University Pl., Op.
CARSON, W. E.....Jenkins Arcade Bldg., Op.
CONWAY, JOHN M.....5151 Jenkins Arcade, ALR
CURRY, GLENDON E.....Westinghouse Bldg., Op.
DAY, KENNETH M.....121 University Pl., ALR
DAY, EWING W.....121 University Pl., OALR
FISCHER, N. A.....623 Union Trust Bldg., ALR
GROSS, JULIUS E.....8030 Jenkins Arcade Bldg., ALR
GUERINOT, A. J.....604 Westinghouse Bldg., OALR
HAGEMANN, JOHN A.....Highland Bldg., ALR
HAMPSEY, A. R.....806 May Bldg., ALR
HECKEL, EDWARD B.....Jenkins Arcade Bldg., Op.
JAQUISH, CHARLES J.....904 Westinghouse Bldg., ALR
KNEEDLER, G. C.....Professional Bldg., ALR
KREBS, ADOLPH.....Jenkins Arcade Bldg., Op.
LINN, J. G.....7026 Jenkins Arcade Bldg., Op.
MCCREADY, J. H.....121 University Pl., ALR
MCKEE, GEORGE J.....Westinghouse Bldg., ALR
MACLACHLIN, A. A.....May Bldg., ALR
MARKEL, J. CLYDE.....Westinghouse Bldg., Op.
MARSHALL, WATSON.....121 University Pl., ALR
MEANOR, WM. C.....619 Westinghouse Bldg., Op.
PATTERSON, ELLEN J.....121 University Pl., ALR
PLUMER, JOHN S.....121 University Pl., Op.
SEEGMAN, SIMON.....Medical Bldg., ALR
SHUMAN, G. H.....Park Bldg., Op.
SIMPSON, JOHN REID.....825 Highland Bldg., ALR
SMITH, STANLEY.....121 University Pl., Op.
STIEREN, E.....626 Union Trust Bldg., Op.
STURM, S. A.....7036 Jenkins Arcade Bldg., Op.
TAYLOR, M. C.....1024 May Bldg., Op.
TODD, GROVER CLEVELAND.....Jenkins Arcade Bldg., ALR
TURNER, H. H.....902 Professional Bldg., Op.
WEISSER, E. A.....806 May Bldg., Op.
YOST, WALTER MILTON.....Westinghouse Bldg., Op.

READING

LANDIS, JAMES E.....517 Walnut St., ALR
RHODE, SOLON L.....238 N. 6th St., OALR
STOCKLER, J. A.....332 N. 5th St., OALR

RIDGWAY

MCALLISTER, J. C.....10 S. Mill St., AL

ROCHESTER

YOST, WALTER M.....Peoples Bank Bldg., Op.

SAYRE

WEINBERGER, NELSON S.....Robert Packard Hosp., OALR

SCRANTON

CORSER, J. B.....345 Wyoming Ave., Op.
CURTIN, EUGENE A.....703 Connell Bldg., OALR
SULLIVAN, J. J., JR.....228 Scranton Life Bldg., ALR
WILLIAMS, M. M.....Medical Arts Bldg., Op.

SUNBURY

BECKER, H. M.....49 S. 4th St., OALR

WARREN

BALL, MICHAEL V.....316 Hazel St., OALR

WASHINGTON

MCCULLOUGH, CLARENCE JOSEPH.....Washington Trust Bldg., ALR
MCKENNAN, JAMES W.....514 Washington Trust Bldg., Op.
MCMURRAY, J. B.....Washington Trust Bldg., OALR

WAYNESBURG

STAHLMAN, FRED C.....Commercial Bldg., OALR

WILKES-BARRE

BUCKMAN, E. U.....70 S. Franklin St., OALR
CARR, GEORGE W.....54 S. Franklin St., OALR
KISTLER, O. F.....43 N. Franklin St., Op.AR
MCLAUGHLIN, P. A.....71 S. Washington St., OALR
MARSDEN, W. C.....64 N. Franklin St., ALR
SCHAPPERT, N. L.....59 S. Washington St., OALR

YORK

FACKLER, CHAS. L.....11 E. Market St., ALR
KLINEDINST, JOHN FERD.....220 S. George St., Op.A

RHODE ISLAND

NEWPORT

JACOBY, D. P. A.....106 Touro St., Op.AL

PROVIDENCE

ADAMS, FRANK M.....122 Waterman St., ALR
BOLOTOW, NATHAN A.....108 Waterman St., ALR
CAPRON, F. P.....118 Angell St., OALR
HARVEY, N. D.....112 Waterman St., OALR
LEECH, J. W.....369 Broad St., OALR
MESSINGER, H. C.....210 Angell St., Op.
MUNCY, WM. M.....162 Angell St., OALR

PORTER, L. B.....199 Thayer St., OALR
SHARP, BENJ. S.....339 Thayer St., OALR
TINGLEY, LOUISA PAINE.....171 Westminster St., Op.

SOUTH CAROLINA

CHARLESTON

KOLLOCK, C. W.....86 Wentworth St., OALR
TOWNSEND, JOHN FERRARS.....85 Wentworth St., OALR

GREENVILLE

CARPENTER, E. W.....Professional Bldg., OALR
MAULDIN, L. O.....Chamber of Commerce Bldg., OALR

SPARTANBURG

GRAY, E. B.....909 Montgomery Bldg., ALR

SOUTH DAKOTA

CHAMBERLAIN

FARNSWORTH, M. A.....Chamberlain Sanitarium and Hospital, OALR

HOT SPRINGS

DEAN, ARTHUR CLARK.....115 River Ave., ALR

HURON

GROSVENOR, L. N.....366 Dakota Ave. So., OALR

MITCHELL

BOBB, E. V.....101 First Nat'l Bank Bldg., OALR

RAPID CITY

WALSH, JOSEPH MARK.....OALR

SIOUX FALLS

GREGG, JOHN B.....612 Security Nat'l Bank Bldg., OALR
KELLER, S. A.....Sioux Falls Clinic, OALR

WATERTOWN

JOHNSON, A. E.....OALR

YANKTON

HOHF, JULIUS.....Clinic Bldg., OALR

TENNESSEE

CHATTANOOGA

HACKNEY, F. J.....630 Volunteer Bldg., OALR
HOGSHEAD, J. M.....602 Georgia Ave., OALR
LAWWILL, STEWART.....Medical Arts Bldg., OALR
STEELE, WILLARD.....Provident Bldg., OALR

DYERSBURG

BREWER, JOHN D.....Baird Brewer General Hospital, OALR

JACKSON

DANCY, ALEXANDER B.....First National Bank Bldg., OALR

JOHNSON CITY

JONES, U. G.....Jones Eye, Ear, Nose and Throat Hospital, ALR

KNOXVILLE

KINCAID, J. H.....421 W. Church Ave., OALR
POTTER, W. W.....Medical Bldg., OALR
REAVES, ROBERT G.....422 W. Cumberland Ave., OALR

MEMPHIS

ANTHONY, DANIEL HARBERT.....1720 Exchange Bldg., Op.
BLASSINGAME, C. D.....899 Madison Ave., ALR
BLUE, J. B.....Columbian Tower OALR
ELLETT, E. C.....Exchange Bldg., Op.
FARRINGTON, P. M.....Phys. and Surg. Bldg., ALR
HUGHES, J. A.....Sterick Bldg., OALR
LEVY, LOUIS.....1052 Madison Ave., ALR
LEWIS, A. C.....Exchange Bldg., OALR
LEWIS, PHILIP MERIWETHER.....Exchange Bldg., OALR
MCKINNEY, R.....899 Madison Ave., ALR
MINOR, H. F.....Randolph Bldg., OALR
PAYNE, VIRGIL LEROY.....1018 Madison Ave., ALR
QUALLS, HARLEY W.....Exchange Bldg., OALR
RYCHENER, RALPH O.....1720 Exchange Bldg., Op.
SELIGSTEIN, MILTON B.....801 Union and Planters Bank Bldg., OALR
SHEA, J. J.....1018 Madison Ave., ALR
SIMPSON, W. LIKELY.....Phys. and Surg. Bldg., ALR
STANFORD, J. B.....1052 Madison Ave., OALR
STINSON, WM. DAVID.....805 Medical Arts Bldg., ALR
WATKINS, EDWIN DIAL.....1412 Exchange Bldg., OALR

NASHVILLE

BRYAN, JAMES LESLIE.....919 Bennie-Dillon Bldg., OALR
CULLOM, M. M.....1119 Bennie-Dillon Bldg., OALR
EZELL, H. C.....228 Doctors Bldg., OALR
HASTY, F. E.....Medical Arts Bldg., ALR
KENNON, W. G.....Doctors Bldg., OALR
WARNER, ROBERT JAY.....Doctors Bldg., Op.

TEXAS

AMARILLO

DUNCAN, ROBERT AUSTIN.....339 Amarillo Bldg., ALR

AUSTIN

BOERNER, MORRIS H.....Norwood Bldg., OALR
HILGARTNER, H. L.....209 Norwood Bldg., Op.
KEY, SAM N.....Norwood Bldg., OALR

BROWNWOOD

ANDERSON, W. B.....Medical Arts Hospital, OALR

CORPUS CHRISTI

STROUD, EDWARD F.....Medical Professional Bldg., ALR

DALLAS

CARPENTER, E. R.....Medical Arts Bldg., OALR
CARY, E. H.....Medical Arts Bldg., OALR
DECHERD, H. B.....1515 Medical Arts Bldg., OALR
McLAURIN, J. G.....Medical Arts Bldg., OALR
McREYNOLDS, JOHN O.....907 Mercantile Bank Bldg., OALR
NEWTON, F. H.....907 Mercantile Bank Bldg., OALR
NELSON, LEO A.....4105 Live Oak St., ALR
SEAY, D. E.....907 Mercantile Bank Bldg., OALR
SCHULZE, E. C.....Medical Arts Bldg., OALR
SELLERS, L. M.....Medical Arts Bldg., ALR
STOKES, WM. H.....4105 Live Oak St., Op.

EL PASO

IRVIN, EDGAR H.....903 First Nat'l Bank Bldg., OALR
SCHUSTER, FRANK P.....403 First Nat'l Bank Bldg., OALR
SCHUSTER, S. A.....2000 N. Mesa Ave., ALR

FORT WORTH

HOWARD, EDWARD L.....405 Medical Arts Bldg., OALR
JACKSON, ATRAS E.....Medical Arts Bldg., Op.
LEA, JOSEPH A.....3526 Travis St., ALR
RICHARDSON, JAMES JOSEPH.....Cook Hosp. and Beall Clinic, ALR
SCHENCK, C. P.....Medical Arts Bldg., OALR
WARWICK, HAROLD L.....913 Medical Arts Bldg., ALR

GREENVILLE

WARD, JAMES W.....Exchange Bank Bldg., ALR

HOUSTON

CODY, CLAUDE C., JR.....1304 Walker Ave., ALR
DAILY, LOUIS.....1117 Medical Arts Bldg., OALR
DAILY, RAY K.....1117 Medical Arts Bldg., OALR
FOSTER, J. H.....1304 Walker Ave., ALR
GOAR, EVERETT LOGAN.....1300 Walker Ave., Op.
GRIFFEY, EDWARD W.....1300 Walker Ave., ALR
HADEN, HENRY C.....1914 Travis St., Op.
ISRAEL, NORMA E.....2001 Niels Esperon Bldg., OALR
ISRAEL, SIDNEY.....2001 Niels Esperon Bldg., ALR
RALSTON, WALLACE.....1300 Walker Ave., Op.

LONGVIEW

HURST, V. REEVES.....107 E. Methvin St., OALR

LUBBOCK

MALONE, F. B.....Lubbock Sanitarium, ALR

PAMPA

LANDES, GEORGE A.....OALR

SAN ANGELO

WINDHAM, ROBERT EDWARD.....234 W. Beauregard Blvd., OALR

SAN ANTONIO.

ATKINSON, D. T.....827 Medical Arts Bldg., OALR
BURLESON, J. H.....Eye, Ear, Nose and Throat Hospital, OALR
GILL, WM. DAVIS.....323 Medical Arts Bldg., OALR
GWINN, G. E.....704 Central Trust Bldg., OALR
PARRISH, ROBERT E.....Medical Arts Bldg., ALR
ROSEBROUGH, FRANK HENRY.....Moore Bldg., Op.

TEMPLE

McDAVITT, BERTHA SCHENCK STECKER
Woodson Eye, Ear, Nose and Throat Hospital, ALR
McREYNOLDS, G. S.....Professional Bldg., ALR
WOODSON, BURBANK P.
Woodson Eye, Ear, Nose and Throat Hospital, OALR

TEXARKANA

FULLER, T. E.....100 West Broad St., OALR
MANN, R. H. T.....State Nat'l Bank Bldg., OALR

TYLER

VAUGHN, E. H.....Citizens Nat'l Bank Bldg., OALR

WACO

AYNESWORTH, H. T.....Medical Arts Bldg., OALR
SPENCER, SHELBY CRUTHIRDS.....605 Columbus Ave., Op.

UTAH

OGDEN

HETZEL, C. C.....Eccles Bldg., OALR

PROVO

MERRILL, H. G.....35 East 2nd South St., OALR

SALT LAKE CITY

DONOHER, WM. D.....Boston Bldg., OALR
HAMPTON, ROBERT R.....804 Boston Bldg., ALR
LE COMPTE, E. D.....627 Boston Bldg., OALR
NEHER, EDWIN MANSON.....Boston Bldg., Op.
SMITH, WM. LEROY.....Medical Arts Bldg., Op.
STAUFFER, FRED.....Medical Arts Bldg., OALR

VERMONT

BARRE

JARVIS, DE FOREST C.....107 N. Main St., ALR

BURLINGTON

ARNOLD, FRANCIS J.....182 Pearl St., OALR

RUTLAND

MARSHALL, GEORGE GUERIN.....Gryphon Bldg., OALR

VIRGINIA

HARRISONBURG

PRESTON, HENRY G.....Professional Bldg., ALR

RICHMOND

GATEWOOD, E. T.....Professional Bldg., ALR
HILL, EMORY.....Professional Bldg., Op.
HUGHES, T. E.....1000 W. Grace St., ALR
MILLER, CLIFTON M.....Stuart Circle Hospital, OALR

ROANOKE

GARRETT, J. R.....Mt. Trust Bldg., OALR
GILL, E. G.....711 S. Jefferson St., ALR
HAMLIN, FRED EUGENE.....Shenandoah Life Bldg., ALR
STONE, HARRY B.....Medical Arts Bldg., ALR
YOUNG, CHARLES A.....Medical Arts Bldg., Op.

UNIVERSITY

WOODWARD, FLETCHER DRUMMOND.....Box 162, ALR

WINCHESTER

MCGUIRE, H. H.....105 N. Braddock St., OALR

WASHINGTON

ABERDEEN

MAXEY, E. E.....609 Becker Bldg., OALR

BELLINGHAM

HOWE, SPENCER S.....Bellingham Nat'l Bank Bldg., ALR
VAN KIRK, FRANK J.....Bellingham Nat'l Bank Bldg., OALR

EVERETT

MURPHY, ALBERT B.....2830 Grand Ave., ALR

SEATTLE

ADAMS, FRED'K W.....Roosevelt Clinic, 1114 Boylston Ave., OALR
BELL, WILL OTTO.....1431 Medical-Dental Bldg., Op.
CHASE, E. FRANK.....1008 Cobb Bldg., OALR
DOWLING, J. T.....1115 Terry, OALR

HARTER, JOHN HOWARD ..... Stimson Bldg., ALR
HOFFMAN, W. F. ..... 817 Summit Ave., Op.
MCCOY, L. L. ..... 817 Summit Ave., Op.
SEELYE, W. K. ..... 1215 Fourth Ave., OALR
STILLSON, H. ..... Seabord Bldg., OALR
WURDEMANN, H. V. ..... 320 Stimson Bldg., Op.

SPOKANE

CODD, ALPHONSO N. ..... Paulsen Med. and Dental Bldg., ALR
ROTT, O. M. ..... Paulsen Med. and Dental Bldg., OALR
SMITH, CARROLL L. ..... Paulsen Med. and Dental Bldg., OALR
VEASEY, C. A., SR. ..... Paulsen Med. and Dental Bldg., OALR
VEASEY, C. A., JR. ..... Paulsen Med. and Dental Bldg., OALR

TACOMA

CAMERON, WM. G. ..... Rhodes Medical Arts Bldg., OALR
DAVIES, J. S. ..... Washington Bldg., OALR
HOWE, A. W. ..... Medical Arts Bldg., OALR
PAUL, WM. C. ..... Washington Bldg., ALR
WHEELER, ERNEST C. ..... Townsend Bldg., OALR

WALLA WALLA

GOWEN, GEORGE ROBERT ..... 306 Baker Bldg., ALR

WEST VIRGINIA

BLUEFIELD

ST. CLAIR, C. T. ..... Corner Bland and Ramsey Sts., ALR

FAIRMONT

JOHNSON, H. R. ..... Masonic Temple, American Bldg., OALR

HUNTINGTON

MOORE, T. W. ..... 1050 Fifth Ave., OALR

MORGANTOWN

WYLIE, C. B. ..... 269 High St., OALR

WHEELING

FAWCETT, I. ..... 75 Twelfth St., OALR
JONES, EDMUND LLOYD ..... Wheeling Clinic, OALR
TOMASSENE, R. A. ..... Wheeling Bank and Trust Bldg., OALR
ZUBAK, MATTHEW F. ..... Citizens Mutual Trust Bldg., OALR

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